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EN BEDRIJFSWETENSCHAPPEN

KU LEUVEN

PARTICIPATION IN PREVENTIVE CARE PROGRAMS: INDIVIDUAL DETERMINANTS, SOCIAL INTERACTIONS AND PROGRAM DESIGN

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tot het behalen van de graad
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Nicolas Bouckaert

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Supervisor:	Prof. Dr. Erik Schokkaert
Members:	Prof. Dr. Geert Dhaene
	Prof. Dr. Erwin Ooghe
	Prof. Dr. Magne Mogstad
	Prof. Dr. Tom Van Ourti

Dankwoord

"Life is like topography;
there are summits of happiness and success,
flat stretches of boring routine
and valleys of frustration and failure."
– Calvin & Hobbes, Bill Waterson –

Wie graag in één zin wil weten wat een doctoraat schrijven inhoudt, kan – wat mij betreft – in het citaat hierboven simpelweg het woord 'life' vervangen door 'PhD'. Het is een langdurig werk van bloed, zweet en tranen, maar wel één om fier op te zijn. Slechts een deel van al dat werk is zichtbaar – in de eerste plaats het doctoraatsboek dat je aan het lezen bent – de rest gaat verborgen in uren van lezen, programmeren, schrijven en nadenken; een heel individueel leerproces.

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Contents

General introduction

Strongly advocated by some, rejected by others, preventive care has been a hot topic in public health discussions. Even though preventive health care accounts for only a minor part of the health care budget of most countries – it amounts between 1% and 4% of total health care expenditures in most OECD countries (OECD Health Data, 2013) – we have seen a steady increase in large scale preventive care programs, both in developed and developing countries. Examples thereof are HIV campaigns, deworming drugs programs, children vaccination programs or bed nets against malaria in developing countries and influenza campaigns or cancer screening and vaccination programs in developed countries.

In this doctoral research, we will not examine the sense or nonsense of preventive care, since we would enter the domain of evidence-based medicine. We note that the benefits of certain types of cancer screening are debated or evidence about health improvements remains inconclusive (see e.g. Bach et al. (2012), Djulbegovic et al. (2010), Marmot et al. (2013), Oakley Jr & Johnston Jr (2004), Reade et al. (2013), Smith et al. (2010)). We focus on existing medical preventive care programs.[1] Because of externalities (e.g. in the prevention of communicable diseases) or the program cost-benefit ratio, prevention programs require high participation rates. In the United States, the Centers for Disease Control and Prevention have set clear participation objectives – next to quality targets – which are measured and evaluated over time (National Center for Health Statistics, 2012). For example, the 2010 participation target for cervical cancer screening in the past 3 years for women aged 18 or more is set at 90%; the target for breast cancer screening in the past 2 years for women aged 40 or more is set at 70% and the target for influenza vaccination is set at 90% of the over-65. Similarly, European countries define participation targets for their large scale prevention programs. First of all, we observe that participation rates rarely meet – let alone exceed – the desired targets, even for prevention measures that are generally considered to be cost-effective such as influenza vaccination for the elderly (Nichol, 2003). Secondly, participation in prevention is not equal among all

[1] Medical preventive care is used as opposed to lifestyle choices such as diet or physical exercise.

targeted individuals and might give rise to health disparities by socioeconomic status, by education level, by geographic location, by sex or by other dimensions. The individual's decision to participate (or not) in preventive care programs is the main thread throughout our research project.

The individual participation decision has been studied in the literature, both theoretically and empirically. The seminal paper of Moffitt on non-take-up behavior in means tested benefits serves as a starting point. More recently, take-up of (preventive) health care services has been studied in the health economics literature (e.g. Byrne & Thompson, 2001; Etner & Jeleva, 2013; Howard, 2005; Maurer, 2009; Picone, Sloan & Taylor, 2004; Schmitz & Wübker, 2012; Whynes et al., 2007; Wu, 2003). In economic models, private incentives, i.e. broadly defined costs and benefits, are generally considered the main drivers of the preventive care participation decision. Participation benefits include reduced mortality and morbidity. Participation costs include financial, non-financial, social and psychological costs. Empirical applications relate the participation decision to individual characteristics and beliefs.

Private incentives are important determinants of preventive care participation. We discuss them mainly in the first chapter of this PhD. However, a broader understanding of non-participation behavior should equally include social interaction effects and policy design. The former relates to direct non-market interactions between individuals. The argument is that the individual's participation decision relates to the participation decision of others in the individual's peer group. The fact that other people participate or do not participate can have a social spillover effect. Kremer & Miguel (2007) state that social effects can result from imitating behavior, social learning about e.g. how to use certain medication efficiently, social learning about the benefits and costs of preventive programs and epidemiological externalities. Depending on the dominating driving force, peer effects can be positive or negative. The policy design of a preventive care program is a second element that might affect the participation decision. Differences between programs may arise from eligibility rules, information campaigns, invitation procedures, the technology that is used, the level of organization, delays in enrollment or delays in learning about the screening results... By deciding upon the design of the prevention program, policy makers endogenously influence take-up rates, nudging people's behavior into a certain direction. Most policy reforms operate on an individual's private incentives by altering prices and (non-) monetary costs and benefits. In addition, this might indirectly generate social interactions effects. Understanding how these social interactions influence individual behavior is important for policymaking since they could reinforce or offset the policy

effects on the individual's private incentives to take-up preventive care. Social interaction effects might therefore lead to higher or lower participation rates than otherwise expected and a social program might reach non-targeted individuals and households through social spillovers. On the other hand, policy interventions, such as mass-media campaigns, can also aim directly at changing social norms and social interaction transmission mechanisms. It is clear that private incentives, social interaction effects and policy design are interrelated and difficult to disentangle empirically. A carefully designed empirical strategy is necessary. This identification challenge will be taken up in chapters 2 and 3. Let us now discuss more into detail to the different chapters.

Chapter 1. Differing types of medical prevention appeal to different individuals (joint work with Erik Schokkaert)

In chapter 1, we analyze participation in medical prevention with an expected utility model that is sufficiently rich to capture diverging features of different prevention procedures and disorders. We distinguish primary and secondary prevention for both fatal or non-fatal diseases. Moreover, we introduce a flexible relationship between the specific disease for which the prevention procedure is set up and the general background health of the individual.

We derive four main hypotheses from the theoretical model. First, current health is positively related to participation in prevention for fatal diseases (e.g. cancer) and negatively for diseases in which good current health mitigates the effects of the disease (e.g. the flu). Second, mortality risk, future costs and benefits only matter for fatal diseases. Third, decreases in program complexity and prevention costs positively correlate with participation for all disease and prevention types. Fourth, increases in current income positively affect participation for fatal diseases, but the income effect can be either positive or negative for non-fatal diseases. These hypotheses are analyzed empirically using European wide data of the Survey of Health, Ageing and Retirement in Europe (SHARE). We look at six types of preventive care (mammography, dental caries screening, influenza vaccination, blood pressure screening, blood sugar screening, cholesterol screening). The observed correlations provide support for the theoretical predictions.

Chapter 2. Neighborhood peer effects in the use of preventive care

Individual participation in preventive care may depend on preventive health behavior in an individual's peer group. Chapter 2 analyzes the effects of policy changes and social interactions in preventive care participation in the context of new social policies (PROGRESA[2]) in Mexico that aim at encouraging preventive care. The program is targeted at the extreme poor in rural areas and is designed as a conditional cash transfer program, meaning that families receive cash transfers conditional on the household engaging in a set of behaviors. Program requirements include, amongst others, participation in various types of preventive care, such as child growth monitoring, child immunization, blood pressure tests, usage of deworming drugs and cervical cancer screening. We follow the promising approach of analyzing social interactions in real world peer groups. Identification of social interactions is based on a partial-population design.

Results indicate that PROGRESA succeeded in increasing preventive care usage among program eligible households. In addition, endogenous social interactions increase preventive care usage for various types of prevention. The magnitude of the effects differs across prevention types. Effects are especially pronounced for annual child growth and weight monitoring. The overall treatment effect of PROGRESA on prevention can be decomposed in a direct effect related to financial incentives and an indirect effect related to social interactions. The indirect effect accounts for 10% up to 60% of the total treatment effect.

Chapter 3. Unintended spillover effects of influenza vaccination: a regression discontinuity approach.

In chapter 3, we investigated direct and spillover effects of an extension of the target group for the Dutch influenza vaccination program to all Dutchmen aged 65 years and over in 1996. Members of the target group qualify for free influenza vaccination and receive a personal invitation letter from their GP. Using a rich dataset that combines survey data on health with administrative records from Statistics Netherlands, the quasi-random variation that was introduced at age 65 by the reform is exploited to analyze vaccination behavior and its impacts on the arguably even more important outcomes of morbidity, medical care use, sickness absence and mortality. While the effects on the

[2]PROGRESA is an acronym for Programa de Educacion, Salud y Alimentacion (the Education, Health and Nutrition Program).

targeted population are useful to evaluate direct policy effect, our primary aim is to estimate policy induced spillovers onto non-targeted individuals, in our setting the adult children of targeted individuals.

Our results indicate a positive direct policy effect on vaccination coverage of the parents (an increase in vaccination rates from about 30% to 50%), accompanied by a negative spillover effect from parents to children (a decrease in vaccination rates from about 9% to 5%). In addition, we estimate that the influenza vaccination program saves 0.8 individuals out of 100,000 at the age threshold, and reduces the number of individuals consulting a GP and using prescribed medicines with 10 percentage points during the typical influenza months. Mortality and GP visits of the adult children are not affected, but the occurrence of influenza-like symptoms increases from 45% to 55% and sickness absence among this group increases from 14% to 22%. We explore several possible channels that might generate the negative spillover effects and find suggestive evidence that a social stigma costs is revealed to children – who are not targeted by the vaccination program – when their oldest parent crosses the age threshold. A potential trigger for the social stigma cost is the explicit framing of the target group in the invitation letter sent out to eligible parents. Our results also underline the importance of public health campaigns to pay attention to the effects of information dissemination on public perceptions and attitudes on (voluntary) preventive care participation.

Chapter 1

Differing types of medical prevention appeal to different individuals

Joint work with Erik Schokkaert

1.1 Introduction

Medical prevention, e.g. vaccination and screening, has become increasingly important in the health care systems of advanced countries. Health practitioners are concerned about the relatively low participation rates, even for prevention measures that are generally considered to be cost-effective (such as influenza vaccination for the elderly and breast cancer screening for women between 50 and 69 years old). A careful look at this participation pattern reveals huge interindividual and intercountry differences. Moreover, participation also varies widely between different procedures for the same individuals. Gaining a better understanding of the causes of these differences across individuals and types of prevention is definitely relevant from a policy point of view.

However, the importance of analyzing medical prevention decisions goes beyond the policy aspect. The large degree of interindividual variation also makes it an interesting domain to apply the theory of decision-making under uncertainty. Our main contribution to the literature is that we integrate existing evidence on participation in medical prevention. Rather than focusing on one specific procedure, our aim is to build and test a model that is sufficiently rich so as to give some insights into the different results that are found with respect to different prevention procedures. We analyze the individual's decision to participate in prevention and how it is affected by the type of prevention offered and the disease characteristics. First, we compare in the same model both primary and

secondary prevention. The former refers to interventions that aim at avoiding or reducing the occurrence of a disease (e.g. vaccination), and the latter to measures that aim at reducing the health consequences of a disease by detection and treatment in its early stages (e.g. cancer screening). Second, we distinguish between fatal (e.g. cancer) and non-fatal (e.g. dental caries) diseases. Third, we introduce into our model a flexible relationship between the specific disease for which the prevention procedure is set up and the general background health of the individual. In some cases, individuals may care more about the specific disease when their background health is worse (e.g. influenza), in other cases they may care more when their background health is better (e.g. dental caries). We show how these various possibilities change the comparative statics of the prevention decision and test the differential predictions with data from SHARE (Survey of Health, Ageing and Retirement in Europe).

We stay in the tradition of the expected utility-approach to study individual preventive medical behavior (see, amongst others, Dervaux and Eeckhoudt, 2004; Picone et al., 2004; Howard, 2005; Witt, 2008). The expected utility-model has recently come under sharp criticism. It is now widely accepted that it is unable to explain real-world observations if one assumes a narrow specification of utility (e.g. focusing only on health and income) and perfectly informed individuals. Taking a test imposes not only monetary (and time) costs, but also a psychological burden, which, according to the available surveys on motivations, may be crucial in explaining variations in preventive care participation (see, e.g., Whynes et al., 2007). Moreover, while the literature has shown that subjective probabilities influence individual decisions, it has also become clear that the subjective risk perceptions vary only very partially with objective risk factors (Carman and Kooreman, 2011). Therefore, the expected utility model only makes sense as an explanation of behavior if all variables used in the model are individual-specific or an individual-specific interpretation of an objective parameter. This is acknowledged by most authors in the field, and we also adopt this interpretation.

In our empirical work we focus on six cases: breast cancer screening, dental caries screening, influenza vaccination, cholesterol screening, blood pressure screening and blood sugar screening. These six procedures cover the range of interesting possibilities suggested by our model. We estimate probit models with the pooled data of the first two waves of SHARE. There have been previous empirical studies analyzing partly the same prevention procedures with SHARE data (Maurer, 2009; Schmitz and Wübker, 2011 and Jusot et al., 2012 for influenza vaccination; Wübker, 2012a, 2012b and Jusot et al., 2012, for mammography; Listl, 2011 and Listl et al., 2012 for dental care). To the best of our

knowledge, we present the first attempt to compare the results for the different procedures within a coherent theoretical approach, testing specific hypotheses about the differential comparative static effects. In accordance with the estimation strategies in Wübker (2012a) for breast cancer screening and Listl et al. (2012) for dental care, we explain (part of) the intercountry differences through the introduction of institutional features that are specifically related to the prevention procedures analyzed. These specific features can be related to the parameters from our theoretical model. This approach appears more promising than controlling for general characteristics of a country's health care system (Jusot et al., 2012[1]).

The remainder of chapter 1 is structured as follows. Section 1.2 describes our model with different types of disorders and characteristics of the process of medical prevention. Comparative static results for the prevention decision are derived in Section 1.3. Section 1.4 discusses the empirical testing of the hypotheses that are derived from the theoretical model. In general the performance of the model in explaining the differences between the procedures is very satisfactory and robust to different specifications. Section 1.5 concludes.

1.2 Model of medical prevention

We propose an expected utility (EU) model that captures an individual's decision to participate in medical prevention for a specific disorder. Participation in prevention is taken to be a binary decision and is pursued when the expected utility of participation exceeds the expected utility of non-participation, i.e. $\Delta EU > 0$, with

$$\Delta EU = EU^{participation} - EU^{non-participation} \tag{1.1}$$

Eq. (1.1) presents the individual decision model in its most simplified form. In what follows, we further detail the expected utility model as both participation and non-participation in prevention may lead to multiple potential health states characterized by a probability and a utility pay-off. Rather than specifying a continuous value for the severity and survival rate of a disorder, we define several disease development stages and a clear, binary distinction between fatal and non-fatal diseases. This simplification with respect to disease characteristics allows, on the one hand, for a richer specification on the characteristics of the individual and the type of prevention, and, on the other hand, does

[1]None of the general characteristics used in this article turn out to have a significant effect for the explanation of influenza vaccination and breast cancer screening.

not unnecessarily complicate the interpretation of the results. We will first work out the model for secondary prevention. Then, we will discuss primary prevention and show how it fits into the same model.

1.2.1 Secondary prevention

During each period t[2], the individual derives utility $u(.)$ from income y, general background health represented by an index h and prevention-specific health m. Utility is concave in income, with $u_1(y,h,m) > 0$ and $u_{11}(y,h,m) \leq 0$.[3] A better general background health corresponds to a higher index score h with $u_2(y,h,m) > 0$ and $u_{22}(y,h,m) \leq 0$. Variable m represents the severity of the specific medical disorder for which the prevention procedure is set up. It takes one of four discrete values ($0 < e < l < d$), ranging from 0, i.e. the individual does not suffer from the disorder, to d, the terminal stage of the disorder, in which the disorder cannot be treated anymore. The values e and l indicate early and late stages of the disorder respectively. The stages are mutually exclusive. The individual believes that she will develop the specific disorder with probability p. The out-of-pocket costs of treatment for the individual are c_e and c_l for, respectively, early and late stage treatment. They are independent of the individual's background health. If treated, the patient is cured of the illness, but relapse in a later period remains possible. The prevention behavior of the individual determines whether the disease develops into early or late stage.

Throughout the main analysis, we impose separability between utility from income and from health, i.e. $u(y,h,m) = v(y) + w(h,m)$. This assumption is widely used in the literature.[4] Appendix 1 presents the results for the unrestricted utility function $u(y,h,m)$.

[2] In our model a 'period' is defined as the normal amount of time in which an individual has to choose whether or not to participate in prevention. For influenza, a period is a one-year interval, since an individual will have to decide to participate in prevention every year before the influenza season starts. For breast cancer screening on the other hand, the normal screening interval is two years. Furthermore, we assume for simplicity and clarity that this amount of time corresponds to the period in which a disease can develop into a severe illness that requires curative care, or in case of a fatal disease, that might result in death. While this is true for many diseases such as e.g. influenza, this is not always the case. The assumption can however be relaxed and our model adapted so that the prevention period and the period of disease development do not necessarily coincide. In this section, we drop the subscript t for notational convenience.

[3] We define $u_x(y,h,m)$ as the derivative of $u(y,h,m)$ with respect to the x^{th} argument of $u(.)$. Analogously, $u_{xz}(y,h,m)$ is the cross derivative of $u(y,h,m)$ with respect to the x^{th} and the z^{th} argument of $u(.)$.

[4] Income can be used for consumption goods that are complements to good health, e.g. travel, or substitutes for good health, e.g. assistance with self-care or a guide dog for the blind. The existing empirical results with respect to the sign and the magnitude of the cross-effect between health and income (or consumption) are inconclusive (Finkelstein et al., 2009).

In addition to utility from current income and health, the individual takes into account future utility V_{t+1} which depends on the future streams of income and health. It is discounted with factor β and corrected for the individual's mortality risk ($p_{x,t+1}$) from any other cause but the prevention-specific disorder. The general utility specification that will reappear in each health state is then the following:

$$v(y) + w(h, m) + \beta(1 - p_{x,t+1})V_{t+1} \tag{1.2}$$

Our two-dimensional representation of health allows us to distinguish between three types of specific disorders in terms of their interaction with the general background health status.[5]

Complements Consider first the case of a rather minor medical problem, which does not affect the background health of the individual: dental caries is an obvious example. In this case, it is natural to assume that "quality of the teeth" matters more for healthier individuals. This is represented in our model by

$$w_1(h, m_1) < w_1(h, m_2), \forall h \text{ if } m_1 > m_2 \tag{1.3}$$

Comorbidities An alternative situation is the case of comorbidities, where the occurrence of the disease has a stronger effect on health if background health is worse. A good example is influenza, since a healthy individual will suffer less from it than a sick individual, and runs a smaller risk of complications. If the utility loss due to the disorder is mitigated by a better initial health, this results in

$$w_1(h, m_1) > w_1(h, m_2), \forall h \text{ if } m_1 > m_2 \tag{1.4}$$

Independence In principle it is also possible that the effect of the new disorder is largely independent of the initial overall health status, resulting in

$$w_1(h, m_1) = w_1(h, m_2), \forall h, m_1, m_2. \tag{1.5}$$

Perhaps an extreme diagnosis like that of a life-threatening cancer could be an example of independence, although in many cases comorbidities would be relevant for cancer also.

[5] We define h as a unidimensional indicator. General background health could alternatively be defined as a multi-dimensional concept, in which the different dimensions of h interact with the severity of the specific disease m. This would make the model less tractable without generating important additional insights.

The classification of different diseases in one of the three categories is ultimately an empirical matter.

1.2.1.1 Potential health states in case of non-participation

The default situation is one where the individual does not participate in preventive care. Ex ante, she believes with a probability $1-p$ that she will be healthy and with a probability p that she will be hit by the disorder. In the latter case, the disease will develop to the late stage, and there are two options. Either the disease is non-fatal and can be cured with treatment at a cost c_l, or, the disease is fatal (e.g. certain cancers, or aggressive viral diseases such as Ebola) and cannot be cured, resulting in the individual's death. When the individual dies, we assume that she no longer benefits from current or future income. To that end, we introduce an indicator function $I(nf)$ that equals 1 if a disease is non-fatal and turns 0 for fatal diseases. The expected utility in the non-participation case can therefore be written as

$$EU^{non-participation} = (1-p)\, u^{HE} + pu^{S}, \tag{1.6}$$

where the utilities in the healthy (HE) and sick (S) states are given respectively by

$$u^{HE} = v(y) + w(h,0) + \beta(1-p_{x,t+1})V_{t+1} \tag{1.7}$$

$$u^{S} = I(nf)\left[v(y-c_l) + w(h,l) + \beta(1-p_{x,t+1})V_{t+1}\right] \tag{1.8}$$

1.2.1.2 Potential health states in case of participation

Secondary prevention allows early treatment of the disease ($m = e$) at a lower cost of treatment $c_e < c_l$. Let us take breast cancer screening as an example. In the typical case, mammograms are used as screening technology. There are alternatives, such as self-control of the breasts or examination of the breasts by the general practitioner (GP) or a more invasive breast tissue biopsy. Every screening technique entails different monetary, psychological (e.g. distress), physical (e.g. pain) and transaction costs (e.g. waiting and travel time). On the other hand, prevention can also induce positive emotions such as reassurance or relief. We indicate the intensity of the preventive procedure by $\alpha > 0$, the out-of pocket monetary cost by c_α and the psychic costs and benefits by $f(\alpha)$ (with $\frac{\partial f(\alpha)}{\partial \alpha} > 0$).

In the case of a positive test, the screening technique successfully detects the presence of the disorder. A true negative test rightly shows that an individual does not suffer

from the disorder. In some cases (including that of breast cancer), the first test round is not perfectly accurate. However if the first test is negative, no follow-up test is taken. If the disorder was undetected, individuals can end up in the late or terminal stage of the disorder. This is called a false negative test result.[6] The utility consequences of the different potential health states are as follows:

$$u^P = v(y - c_\alpha - c_e) + w(h, e) + \beta(1 - p_{x,t+1})V_{t+1} - f(\alpha) \tag{1.9}$$

$$u^{TN} = v(y - c_\alpha) + w(h, 0) + \beta(1 - p_{x,t+1})V_{t+1} - f(\alpha) \tag{1.10}$$

$$u^{FN} = I(nf) \times [v(y - c_\alpha - c_l) + w(h, l) + \beta(1 - p_{x,t+1})V_{t+1}] - f(\alpha) \tag{1.11}$$

where the superscripts P, TN, FN refer to "positive", "true negative" and "false negative" respectively.

The probabilities of ending up in the different health states depend on the effectiveness of the prevention program as measured by the test sensitivity ($se \in [0, 1]$). Test sensitivity is defined as the probability that a test will be positive for an ill individual and can be expressed in terms of the numbers of positive (N^P) and false negative tests (N^{FN}):

$$se = \frac{N^P}{N^P + N^{FN}} \tag{1.12}$$

We can then write the probabilities to end up in a certain health state in terms of p and se:

$$p^P = p \times se \tag{1.13}$$

$$p^{TN} = (1 - p) \tag{1.14}$$

$$p^{FN} = p \times (1 - se) \tag{1.15}$$

If we combine the utility pay-off and probabilities, we can formulate the expected utility in case of participation in a preventive care program:

$$EU^{participation} = p \times se \times u^P + (1 - p) \times u^{TN} + p \times (1 - se) \times u^{FN} \tag{1.16}$$

[6] After a positive test result, a more conclusive second test (e.g. breast tissue biopsy) can reveal that the disorder was falsely suggested in the first round while the individual does not have the disorder. This is defined in the literature as a false positive test. The frequency of false positive results is captured by the test specificity, which is the probability that the test yields a negative result for an individual without the disorder. In order to simplify our analysis we abstract from the possibility of a second screening round. The results from a more complete model are similar. They can be obtained from the authors on request.

From the point of view of the patient, test sensitivity se is important for those who are hit by the disease. In utility terms, the relevance of a larger value for se is expressed by the difference between u^P and u^{FN}. If the screening test gives perfect information we have $N^{FN} = 0$ and $se = 1$. In principle, one can expect that the medical prevention technology implies a positive relationship between sensitivity se on the one hand and test intensity α on the other. This relationship need not be monotonic, though.

1.2.1.3 The full model

The individual will participate in prevention if $\Delta EU > 0$, with

$$\begin{aligned}
\Delta EU &= EU^{participation} - EU^{non-participation} \\
&= p \times se \times u^P + (1-p) \times u^{TN} + p \times (1-se) \times u^{FN} \qquad (1.17) \\
&\quad -p \times u^S - (1-p)u^{HE} \\
&= p \times se \times \left[u^P - u^{FN}\right] + (1-p) \times \left[u^{TN} - u^{HE}\right] + p \times \left[u^{FN} - u^S\right] \qquad (1.18)
\end{aligned}$$

It is useful to consider the relative ranking of the different states. For most realistic values of $f(\alpha)$, i.e. if psychological costs and distress due to prevention exceed psychological benefits from reassurance, and preventive care costs are not excessive, it is clear from eqs. (1.7) to (1.11) that $u^{HE} > u^{TN} > u^P$, and that $u^S > u^{FN}$. It is sufficient to assume that $u^P > u^S$ to get a full ranking. For fatal diseases (with $u^S = 0$), this assumption boils down to the innocuous premise that taking an effective preventive action to avoid death yields a positive utility outcome ($u^P > 0$). For non-fatal diseases, we derive from eqs. (1.9) and (1.8):

$$u^P - u^S = v(y - c_\alpha - c_e) - v(y - c_l) + w(h, e) - w(h, l) - f(\alpha), \qquad (1.19)$$

A positive value implies that the utility gain due to early discovery and treatment instead of late treatment is larger than the psychological costs related to prevention. In that case, we can conclude that:

$$u^{HE} > u^{TN} > u^P > u^S > u^{FN} \qquad (1.20)$$

If we accept eq. (1.20), the first term in eq. (1.18) is positive and represents the utility gain from a correct diagnosis and early preventive effort. Increasing sensitivity se leads to more effective prevention, and hence to a utility increase. The second and third term,

on the other hand, are obviously negative. The second term represents the utility loss for a healthy person when participating in prevention. The third term indicates the utility loss due to a wrong screening diagnosis.

There are, however, two extreme cases: always-compliers and never-compliers. Some individuals enjoy prevention and will always comply. This is the case if psychological benefits exceed psychological costs and monetary prevention costs are limited. In this case $u^P > u^{FN} > u^S$ and $u^{TN} > u^{HE}$, so that all three terms in eq. (1.18) turn positive. On the other hand, it is possible that for some individuals, the psychological costs are prohibitively high, so that $u^S > u^P > u^{FN}$. This implies that $p \times u^S > p \times se \times u^P + p \times (1 - se) \times u^{FN}$ and that the expected utility from prevention given by eq. (1.17) is always negative. These individuals never comply with prevention. We exclude the extreme cases from the further analysis.

1.2.2 Primary prevention

Primary prevention (for example immunization or Aspirin use to prevent cardiovascular diseases) reduces the probability of developing a disorder. The aim is not to detect and treat a disease in its early stages, but to keep the individual healthy in the first place. The expected utility for non-participation in prevention is identical to the specification in eqs. (1.6) to (1.8). The expected utility for participation changes, because the potential health states are different.

In case the individual participates in primary prevention, two potential health states exist: the individual can either become sick or remain healthy. The former occurs when the preventive technology is not effective (NE). A sick patient will be referred to late-stage treatment (or will die if the disease is fatal). The utility pay-off and the probability of ending up in this state are:

$$u^{NE} = I(nf) \times [v(y - c_\alpha - c_l) + w(h, l) + \beta(1 - p_{x,t+1})V_{t+1}] - f(\alpha) \quad (1.21)$$

$$p^{NE} = p(1 - ef) \quad (1.22)$$

where ef is a parameter that denotes the effectiveness of the primary preventive technology.

Alternatively, the individual remains healthy when she is not affected by the disease or the preventive technology effectively protected her from contracting the disease. Let

us call this the effective state (E). The utility pay-off and the probability of ending up in this state are:

$$u^E = v(y - c_\alpha) + w(h, 0) + \beta(1 - p_{x,t+1})V_{t+1} - f(\alpha) \quad (1.23)$$

$$p^E = 1 - p(1 - ef) = (1 - p) + p \times ef \quad (1.24)$$

Remark that the expressions are very much in line with the specifications we have used to model secondary prevention. Eqs. (1.21) and (1.23) are identical to eqs. (1.11) and (1.10), respectively. If we reinterpret the sensitivity parameter for secondary prevention as the effectiveness parameter for primary prevention, it is clear that $p^{NE} = p^{FN}$ and $p^E = p^{TN} + p^P$. In fact, the main difference between both types of prevention is that the positive test state with early treatment for secondary prevention is replaced by the true negative state in case of primary prevention. The expected utility for participation can be written as follows:

$$EU^{participation} = [(1 - p) + p \times ef] \times u^E + p(1 - ef) \times u^{NE} \quad (1.25)$$

$$= \left[p^{TN} + p^P\right] \times u^{TN} + p^{FN} \times u^{FN}, \text{ for } ef = se \quad (1.26)$$

From eq. (1.20), we know that $u^{TN} > u^P$. The replacement of u^P by u^{TN} in eq. (1.26), shows that, *ex ante* and *ceteris paribus,* in comparison with secondary prevention, primary prevention makes it possible to realize an additional utility gain of the order $p^P \times \left(u^{TN} - u^P\right)$.

1.3 Comparative statics of the prevention decision

Individuals that do not expect to die in the immediate future will be confronted for a given disease with multiple decision moments to participate in preventive care. The same decision problem will return in the next period and this process continues until the uncertain moment of death. This means that the individual decides whether or not to participate in prevention in the current period, taking into account future utility and future preventive effort. To model this full process, one would need a multi-period model. However, such a multi-period model is mathematically burdensome. We sketch its main features in Appendix 2, but here we focus on a simplified two period model that is sufficient to yield the main insights.

In this simplified model, we assume that the individual lives during two periods and dies at the end of the second period. In period 1, the individual decides whether or not to participate in the preventive program, while in period 2, the individual does not participate in prevention and simply gets utility from income and health. The expected utility in period 2 is unaffected by individual behavior and is characterized as follows:

$$V_2 = (1-p_2) \times [v(y_2) + w(h_2, 0)] + I\,(nf) \times p_2 \times [v(y_2 - c_l) + w(h_2, l)] \tag{1.27}$$

Let us now implement the decision rule eq. (1.1) for the first period. This gives for secondary prevention:

$$\begin{aligned} \Delta EU_1 \;=\;& p_1 \times se \times [v(y_1 - c_\alpha - c_e) + w(h_1, e) + \beta(1-p_{x,2})V_2] \\ & +(1-p_1) \times [v(y_1 - c_\alpha) - v(y_1)] - f(\alpha) \\ & +I(nf) \times \left[\begin{array}{c} p_1 \times (v(y_1 - c_\alpha - c_l) - v(y_1 - c_l)) \\ -p_1 \times se \times (v(y_1 - c_\alpha - c_l) + w(h_1, l) + \beta(1-p_{x,2})V_2) \end{array} \right] \end{aligned} \tag{1.28}$$

and for primary prevention (with $se = ef$):

$$\begin{aligned} \Delta EU_1 \;=\;& p_1 \times se \times [v(y_1 - c_\alpha) + w(h_1, 0) + \beta(1-p_{x,2})V_2] \\ & +(1-p_1) \times [v(y_1 - c_\alpha) - v(y_1)] - f(\alpha) \\ & +I(nf) \times \left[\begin{array}{c} p_1 \times (v(y_1 - c_\alpha - c_l) - v(y_1 - c_l)) \\ -p_1 \times se \times (v(y_1 - c_\alpha - c_l) + w(h_1, l) + \beta(1-p_{x,2})V_2) \end{array} \right] \end{aligned} \tag{1.28'}$$

Except for the first line, expressions (1.28) and (1.28') are identical. We will now derive the comparative statics for the utility difference ΔEU_1 for the effect of the future, of background health and of income. The computations for other parameters can be found in Appendix 4 and will be discussed only briefly.

The future It follows from eqs. (1.28) and (1.28') that

$$\frac{\partial \Delta EU_1}{\partial \beta} = (1 - I(nf)) \times p_1 \times se \times (1 - p_{x,2}) \times V_2 \geqslant 0 \tag{1.29}$$

$$\frac{\partial \Delta EU_1}{\partial p_{x,2}} = -(1 - I(nf)) \times p_1 \times se \times \beta \times V_2 \leqslant 0 \tag{1.30}$$

$$\frac{\partial \Delta EU_1}{\partial V_2} = (1 - I(nf)) \times p_1 \times se \times \beta \times (1 - p_{x,2}) \geqslant 0 \tag{1.31}$$

The future only influences the prevention decision in the case of a fatal disease, i.e. if $I(nf) = 0$. Indeed, with a non-fatal disease, all the relevant consequences occur in the first

period and every health state has the same prospects with respect to the future. For fatal diseases, prevention provides an opportunity to avoid death through early treatment and thus increases the probability to benefit from future utility. Participation in prevention rises as the present value of the utility gain related to prevention, increases. This happens when the level of future utility V_2 or the weight β given to the future increase or the probability of dying from other causes $p_{x,2}$ decreases.

We implicitly assume that an individual has a general background health h_2 in period 2, irrespective of being healthy, treated early or late in period 1. This assumption can be relaxed to have e.g. a lower h_2 when treated late, due to permanent health damage, compared to being treated early or not needing treatment at all. This leads to different utility values of V_2 depending on the potential states in period 1. The consequence is again that the future might matter for non-fatal diseases, and, that the marginal effects (w.r.t. $\beta; p_{x,2}; V_2$) go in the same direction as described for a fatal disease.[7]

Income The partial effect for current income y_1 can be derived from (1.28) for secondary prevention:

$$\begin{aligned}\frac{\partial \Delta EU_1}{\partial y_1} &= p_1 \times se \times v_1(y_1 - c_\alpha - c_e) + (1 - p_1) \times [v_1(y_1 - c_\alpha) - v_1(y_1)] \\ &\quad + I(nf) \times [p_1 \times (v_1(y_1 - c_\alpha - c_l) - v_1(y_1 - c_l)) - p_1 \times se \times v_1(y_1 - c_\alpha - c_l)]\end{aligned} \tag{1.32}$$

and from (1.28') for primary prevention:

$$\begin{aligned}\frac{\partial \Delta EU_1}{\partial y_1} &= p_1 \times se \times v_1(y_1 - c_\alpha) + (1 - p_1) \times [v_1(y_1 - c_\alpha) - v_1(y_1)] \\ &\quad + I(nf) \times [p_1 \times (v_1(y_1 - c_\alpha - c_l) - v_1(y_1 - c_l)) - p_1 \times se \times v_1(y_1 - c_\alpha - c_l)]\end{aligned} \tag{1.32'}$$

The first line in eqs. (1.32) and (1.32') is always positive. The second line is zero for fatal diseases and can be positive or negative for non-fatal diseases. Therefore for fatal diseases the overall income effect is always positive. If the disease is non-fatal, the sign of the overall income effect depends on the relative size of the underlying parameters.

[7] Another assumptions that influences the comparative statics with respect to the future is that the frequency of prevention and the period of disease development coincide. If this is not the case, and e.g. prevention is recommended to be taken yearly while the disorder needs more than a year to develop to the late stage of the disorder, the prevention decision is taken in period 1 and potential curative treatment occurs in period 2. The consequence of this discrepancy is that the future will also matter for a non-fatal disease, and the marginal effects (w.r.t. $\beta; p_{x,2}; V_2$) go in the same direction as described for a fatal disease.

Table 1.1: Taylor conditions for positive income effect of y_1 on participation in period 1

Disease type	**fatal disease**	**non-fatal disease, primary prevention**	**non-fatal disease, secondary prevention**
Taylor condition y_1	always positive effect	$c_\alpha \geqslant p_1 \times se \times c_l$	$c_\alpha \geqslant p_1 \times se \times (c_l - c_e)$

A first order Taylor expansion around y_1 allows us to formulate approximate conditions for $\frac{\partial \Delta EU_1}{\partial y_1}$ to be positive. The results are summarized in Table 1.1.[8] For non-fatal diseases, income will have a positive effect if the (private) monetary costs of participation in prevention (costs for screening or vaccination) outweigh the savings in terms of curative treatment costs. If monetary costs are larger than monetary benefits, this will have a negative effect on the incentives for prevention, and, with a concave utility function, the negative impact will be more pronounced for poorer persons. This explains the positive income effect on participation in prevention. If costs are less than the benefits, an analogous reasoning yields a negative income effect. The conditions in Table 1.1 are easily interpreted. In most realistic cases of secondary prevention we may expect a positive income effect. If, for example, curative treatment and early treatment are equally well covered by health insurance, any monetary cost of prevention, as minor as it might be, leads to a positive income effect. In the case of primary prevention, the conditions for a positive income effect are stricter.

We can also draw conclusions about the effect of y_2 on the expected utility gain of taking a preventive test in period 1. It has a positive effect on participation, but only for fatal diseases. The obvious intuition is that an income increase enhances future utility V_2 and makes actual preventive effort more beneficial.

General background health The comparative static expressions for background health h_1 are given by:

$$\frac{\partial \Delta EU_1}{\partial h_1} = p_1 \times se \times w_1(h, e) - I(nf) \times p_1 \times se \times w_1(h, l) \tag{1.33}$$

for secondary prevention and for primary prevention by:

$$\frac{\partial \Delta EU_1}{\partial h_1} = p_1 \times se \times w_1(h, 0) - I(nf) \times p_1 \times se \times w_1(h, l) \tag{1.33'}$$

[8] The details of the calculations are given in Appendix 3.

The sign of this expression depends heavily on the type of illness and prevention, as well as on the interaction between h and m as laid out in section 1.2. An overview of the different possibilities is given in Table 1.2. Note that these results offer an alternative explanation for the finding of Wu (2003), who found a positive effect of health on participation in breast cancer screening and a negative effect on influenza vaccination. Wu pointed at psychological factors such as fear and anxiety, varying discount rates by health status or differences in GP advice according to health status to explain this discrepancy. Our model provides an easy explanation within the context of a standard expected utility model, based on the type of prevention and the disease characteristics.[9]

Table 1.2: Overview of the expected effect of health on preventive action according to disease and prevention type

Disease type	**fatal disease**	**non-fatal disease**
Complements h **and** m	positive	positive
Comorbidities h **and** m	positive	negative
Independence h **and** m	positive	no effect

The effect of future health on participation in prevention is similar to the effect of future income. A better future background health makes it worthwhile to pursue prevention in the current period in the case of a fatal disease.

Other parameters The comparative statics for the other parameters can be found in Appendix 4. We conclude from the results that participation in prevention is unambiguously increased by lowering complexity (α), monetary (c_α) and psychological $f(\alpha)$ costs of prevention, by lowering early treatment costs (c_e), by enhancing the effectiveness of the preventive technology ($se; ef$). However as an increase in complexity (α) at the same time raises monetary costs (c_α) and improves effectiveness (se, ef), the positive and negative effects on participation should be weighed against each other. An increase in curative (late) treatment costs has no effect on preventive behavior for fatal diseases, since no cure is available, and an ambiguous effect for non-fatal diseases. For non-fatal diseases, the effect will be positive if the preventive technology is effective and the monetary costs of prevention are low, since in this case prevention provides a good alternative to curative treatment. A similar conclusion can be drawn for risk perceptions p_1. They have a positive effect on participation for fatal diseases, but an ambiguous effect for non-fatal diseases, which will be positive for effective preventive procedures with low monetary costs.

[9] Similar arguments are given by Mullahy (1999) and Maurer (2009).

1.4 Empirical analysis

For our empirical illustration, we analyze six types of disorders and their corresponding preventive care options: breast cancer, dental caries, influenza, hypertension, hypercholesterolemia and diabetes. In the next subsection we briefly describe the disorders and summarize the corresponding behavioral hypotheses. We then present the available data used in the empirical analysis. Finally, we present the results.

1.4.1 Setup of the empirical exercise

1.4.1.1 Six procedures

Breast cancer: fatal disease, secondary prevention, comorbidities or independence in health. Breast cancer is the most common cancer among European women. It accounts for almost one in three new cancer cases and one in six cancer deaths. One in nine women develops breast cancer at some point in her life, and this fraction has increased over the years. Although primary prevention is not yet an option, it is possible to detect breast cancer and the chances of survival increase the earlier the cancer is treated. For this reason, many countries have set up a preventive screening program. Given the nature of breast cancer, we assume that late treatment of cancer results in death during the period.[10]

Dental caries: non-fatal disease, secondary prevention, complements in health. For preventive dental care, no government organized large scale preventive care programs exist. Dental policies vary widely across European countries (Widström and Eaton, 2004). The setup of preventive care is as follows. An (asymptomatic) individual visits the dentist preventively (without feeling pain or having dental-related problems). The dentist screens for dental caries and dental plaque. If the dentist observes irregularities action is undertaken. In the case of no prevention or a false negative result, there will be curative treatment of the advanced dental problem.

Influenza: non-fatal disease, primary prevention, comorbidities in health. Influenza vaccination is one of the best-known and most studied examples of primary prevention. Influenza is a very common infectious disease that causes general discomfort for most and death for some. In the latter case, death is often the result of a weakening of

[10] The American Cancer Society (2013) distinguishes between 4 cancer stages. If breast cancer is detected and treated early (stage 1 or 2), the 5 year survival rate is nearly 100%, whereas survival rates drop to 20% if cancer is detected in stage 4.

the immune system caused by influenza and an additional infection, e.g. pneumonia. The Centre for Disease Control and prevention (CDC) in the United States estimates that each year on average 5% to 20% of the population suffers from seasonal influenza, i.e. 5000 to 20.000 infected individuals per 100.000 persons. The death rate from influenza in the period 1976 – 2007 is estimated between 1,4 to 16,7 deaths per 100.000 persons (Thompson *et al.*, 2010). When combining this information, it is clear that the case fatality risk, which is the risk of dying when infected (or $1 - I(nf)$ in our model), is far less than 1%.[11] Therefore, we consider seasonal influenza to be a non-fatal disease.

Since the disease is infectious, immunization brings about positive externalities. Most developed countries provide subsidized vaccination programs for certain vulnerable groups within the population, such as chronically ill individuals or the elderly. In addition to government programs, a number of companies also provide vaccination programs.

Hypertension: non-fatal disease, secondary prevention, comorbidities in health. Hypertension refers to chronic high blood pressure. In the US, 41% and 55% of non-institutionalized individuals aged above 45 or 65, respectively, indicate to suffer from hypertension in 2012 (Blackwell, Lucas & Clarke, 2014). Hypertension by itself is not life threatening and can be controlled and treated through dietary and lifestyle changes and/or medication. In combination with smoking, drinking or other chronic condition, such as hypercholesterolemia and diabetes, the risk of dying from cardiovascular diseases increases strongly.[12] Therefore, we assume comorbidities in health. There are two types of screening. First, individuals with a previous diagnosis of high blood pressure or of a cardiovascular condition are screened to follow closely their health status and evaluate the effects of treatment against hypertension. Second, asymptomatic individuals, i.e. individual without increased risk for hypertension or cardiovascular diseases, have the possibility to opportunistically measure their blood pressure, e.g. as part of a medical check-up by the GP. Our model is relevant for the latter type of screening.

[11] It should be noted however that mortality varies substantially by influenza virus type and age group. Most of the influenza and pneumonia related deaths occur among adults aged 65 or more. Hadler *et al.* (2010) suggest case fatality rates per age group for seasonal influenza of 0,001% – 0,004% in the age group of 0 to 17 year olds, 0,003% – 0,011% for adults between 18 and 64 and 0,11% to 0,44% for those aged 65 or more.

[12] Cardiovascular diseases are the number 1 cause of death in Western countries. In the US, the incidence of heart diseases in 2012 is 18% and 30% for non-institutionalized individuals aged above 45 and 65, respectively (Blackwell, Lucas & Clarke, 2014). The mortality rate over the entire population in 2010 is 0.5% and 1.2%, for individuals aged above 45 and 65, respectively (National Center for Health Statistics, 2014).

Hypercholesterolemia: non-fatal disease, secondary prevention, comorbidities in health. Hypercholesterolemia refers to chronic high levels of cholesterol in the blood. In the US, 45% and 54% of the individuals aged above 45 or 65, respectively, suffers from high cholesterol levels or takes cholesterol-lowering medication between 2009 and 2012 (National Center for Health Statistics, 2014). High cholesterol levels puts an individual at risk for, amongst other, heart diseases. Cholesterol levels can be lowered mainly by medication. There are no direct signs or symptoms of high cholesterol, so that screening, by means of a simple blood test, is required to ascertain its presence. As for hypertension, we focus on opportunistic screening by asymptomatic individuals.

Diabetes: non-fatal disease, secondary prevention, comorbidities in health. Diabetes is a chronic disease that occurs when the pancreas does not produce enough insulin, or when the body cannot effectively use the insulin it produces, leading to high blood glucose levels. High blood glucose levels for prolonged periods cause damage to blood vessels, nerves and other tissues. This can lead to serious health complications such as cardiovascular diseases, stroke, blindness and kidney failure, especially in combination with disturbances in lipid metabolism, hypertension and smoking. The effects of diabetes can be mitigated for a substantial period of time by adjusting the diet, physical exercise, a healthy lifestyle, insulin injections and other medication. In our analysis, we focus on opportunistic screening by asymptomatic individuals.

1.4.1.2 Hypotheses and empirical specification

Participation in prevention is a discrete decision. In our theoretical model we assumed that individual i participates if $\Delta EU_1^i > 0$, with ΔEU_1^i given in eqs. (1.28) and (1.28'). Adding a stochastic component ε_i capturing idiosyncratic factors, missing variables and measurement errors, we can write the probability of participation as

$$P(i\ participates) = P(\Delta EU_1^i + \varepsilon_i > 0) = P(\Delta EU_1^i > -\varepsilon_i).$$

If we assume the random term to be normally distributed, this results in a standard probit model. The comparative static hypotheses about ΔEU_1, as derived in the previous section, can then be rephrased directly as hypotheses on the probability of participation.[13]

[13] Belkar et al. (2006) show that neglecting to distinguish between "aware" and "unaware" individuals may lead to a selection effect. However, they also show that the problem is not very serious if "censoring is modest and positive dependence between awareness and choice is substantial" (p. 44). This is likely to be the case with our data.

To keep track of our theoretical predictions, it may be convenient to look at Table 1.3, summarizing the hypotheses for the empirical cases that will be analyzed.

Table 1.3: Overview of the theoretical hypotheses

Effect on participation in prevention (period 1)	**Disorder***			
	F, SP, Indep. (e.g. breast cancer)	**NF, SP, Compl. (e.g. dental caries)**	**NF, PP, Comor. (e.g. influenza)**	**NF, SP, Comor. (e.g. hypertension, diabetes, hypercholesterolemia)**
Decrease h_1	negative	negative	positive	positive
Increase y_1	positive	ambiguous (likely positive)	ambiguous (likely positive)	ambiguous (likely positive)
Increase $p_{x,2}$	negative	no effect	no effect	no effect
Decrease α, c_α	positive	positive	positive	positive
Increase p_1	positive	ambiguous (likely positive)	ambiguous (likely positive)	ambiguous (likely positive)

* F = Fatal; NF = Non-Fatal; SP = Secondary Prevention; PP = Primary prevention; Compl. = Complements; Comor. = Comorbidities; Indep. = Independence

1.4.2 Data

Individual microdata are taken from SHARE. For breast cancer screening and influenza vaccination, government organized prevention programs exist that differ across European countries. For both disorders, we combine the microdata from SHARE with information from macrosources about the specific features of the prevention programs in the different countries. Table 1.4 gives an overview of the relevant data and shows how they are related to the variables in our theoretical model. Descriptive statistics are provided in Table 1.7 in Appendix 5.

Individual data Our individual data come from the first (2004-2005) and second (2006-2007) wave of the Survey on Health, Ageing and Retirement in Europe (version 2.5.0). SHARE is a micro-dataset, targeted at individuals aged 50 years and over (plus spouses). It covers more than 30,000 non-institutionalized individuals from 14 European countries and Israel. A household is selected in a random procedure, but with the specific requirement that at least one individual is aged 50 years or over. SHARE provides comparable and detailed individual and household information. A full description can be found in Börsch-Supan et al. (2005).

The dependent variables are binary variables that equal 1 if the individual has had a specific type of prevention in the last (two) year(s). The type of procedures include mam-

Table 1.4: Overview of the data

	Disorder			
Data	**Breast cancer**	**Dental caries**	**Influenza**	**Other disorders**
h_1	– Subjective health status – Objective health variables: ADL, Mobility, BMI, Grip strength			
y_1	– Equivalent household income, broadly defined			
$p_{x,2}$	– Mortality risk over period of 10 years			
α, c_α	– Belonging to country target group for screening – Probability of receiving an invitation letter – Population based program completed		– Belonging to country target group for influenza – Free or subsidized vaccination	
p_1	– Belonging to country target group for screening – Past cancer diagnosis – Age and country specific breast cancer incidence and mortality rates	– Dentures – Trouble biting	- Belonging to country target group for influenza	
Control var.	– Education, Nationality, Gender, Age, Partner, Smoker, House owner, Country dummies by wave			

mograms for women, preventive dental care[14], influenza vaccination, blood cholesterol test, blood pressure test and blood sugar test. Reported participation rates for our subsamples are 55%, 42%, 33%, 52%, 68% and 54% respectively. Despite using two rounds of SHARE, no panel structure can be easily implemented. Data on participation in breast cancer screening and influenza vaccination were collected through a self-administered drop-off questionnaire. For breast cancer screening, we restrict our sample to women without a history of breast cancer. No respondent received the drop-off questionnaire in both waves, therefore we are limited to a pooled cross-sectional analysis for breast cancer screening and influenza vaccination. In the case of dental prevention, an important number of individuals answered the question on participation in preventive dental care in both waves. We account for this by pooling all observations but clustering error terms at the individual level. With respect to blood pressure, cholesterol and sugar tests, individuals are asked whether or not in the past year they were tested by a doctor or a nurse. This question was only surveyed in wave 2 through a self-administered drop-off questionnaire.[15] We focus on opportunistic screening by asymptomatic individuals, and exclude

[14] We set preventive dental care equal to one if individuals reported visting a dentist in the last twelve months for preventive use or prevention and treatment combined. The value is set to zero if the individual has not seen a dentist or has seen them only for treatment. Our empirical results are similar when using an alternative specification with a value equal to one if the dentist is contacted for prevention use only and zero otherwise.

[15] All other SHARE data discussed below were collected using a computer assisted personal interviewing

individuals with heart conditions and diabetes for the subgroup of blood pressure and cholesterol screening. Furthermore, we exclude individuals with a history of hypertension or hypercholesterolemia, respectively for the subgroup of blood pressure and cholesterol screening, as well as individuals who take specific medication for the disorder. For blood sugar screening, we exclude individuals with a history of diabetes or who use medication for diabetics.

As for the explanatory variables, we are particularly interested in variables that allow us to distinguish between the different disease and prevention combinations, i.e. health status and mortality risk. We supplement these with an income variable and various control variables.

SHARE contains subjective and objective health information. Self-assessed health is represented by four dummy variables, reflecting different perceived health levels: poor, fair, good, and, very good/excellent. In addition, there are many variables that capture a part of an individual's general background health in a more objective way. We have two indices: an index of limitations to six activities of daily living[16], and an index of limitations in mobility related to health.[17] For each limitation the individual suffers from, a score of 1 is awarded. Each index represents the sum of all relevant limitations, which is subsequently rescaled (by the total number of potential limitations) to a value between 0 and 1, respectively meaning no limitations and all limitations apply. Furthermore, we have information on the BMI and a score for the grip strength of the individual (ranging from 0 to 100). We use the average score out of 2 measurements with the individual's dominant hand.

The mortality risk ($p_{x,2}$) is captured by the question: "What are the chances that you will live to age X or more?". Age X is predetermined and depends on current age, e.g. for all individuals aged 65 or less, age X is set at 75, for individuals aged between 66 and 70, age X is fixed at 80, etc. Thus, the survival period may differ across individuals and survival probabilities cannot be clearly interpreted. To make mortality risk comparable across individuals, we estimate the probability of dying in the next 10 years using a

(CAPI) program. A self-administered drop-off questionnaire can be biased, since lower socio-economic groups tend to be underrepresented. Therefore, the answers to the drop-off questionnaire might not be representative of the population. However, Jusot et al. (2012) point out that prevalence rates obtained in the drop-off questionnaire correspond to available published OECD population data for most countries.

[16] The activity questions that are used (yes/no): are you able to... dress?, walk across a room?, bathe or shower?, eat?, get in and out of bed? and use the toilet?

[17] The mobility questions that are used (yes/no): Are you able to... walk 100 metres?, get up from a chair after sitting for long periods?, climb stairs?, reach your arms above shoulder level?, carry weights over 5 kilos?

Weibull specification. A Weibull function is frequently used to model longevity (Juckett & Rosenberg, 1993; Wilson, 1994). The CDF of the Weibull distribution has the following form:

$$F(x;k;\lambda) = 1 - e^{-\left(\frac{x}{\lambda}\right)^k} \tag{1.34}$$

with k the shape parameter or death rate, x the time to death, and λ the scale parameter. With the survival probability $(1 - F(x;k;\lambda))$ and two age points, i.e. current age and age X, we can compute x and either λ or k. In absence of additional information on survival probability (e.g. for another time span), we need an assumption on the other parameter. We make assumptions on the death rate, since it is easier to interpret than the scale parameter. $k = 1$ implies a constant death rate at all ages, while $k > 1$ corresponds to an increase of the death rate with age, i.e. older people are more likely to die. Longevity analysis using a Weibull specification suggests a death rate between 4 and 10 for individuals aged between 50 and 100 years (Weon, 2004; Wilson, 1994). Therefore, in our empirical analysis we use $k = 7$ as the baseline death rate and perform a sensitivity analysis for $k \in [4, 10]$. The Weibull specification cannot meaningfully deal with certain survival probabilities of 0% or 100%.[18] However, since nobody can predict survival with certainty, we adjust 'certain' survival probabilities of 0% and 100% to slightly uncertain probabilities of 0.01% and 99.99%. A similar approach has been followed in Picone et al. (2004).[19]

Income is interpreted as equivalent household income (using the square root equivalence scale), comprised of labor and retirement income as well as income from wealth (dividends, rental income etc.). We use reported (not imputed) income data and filter out households with zero or extremely high reported income. All amounts are expressed in euros using the exchange rates provided by SHARE, and subdivided into deciles across the different European countries (separately for each wave).

[18] Our Weiull specification does not allow to compute mortality probabilities for individuals who evaluate survival at 0%, whereas mortality probabilities for individuals who evaluate survival at 100% do not vary with parameter specifications and always give a mortality probability of 0%.

[19] There exists some doubt as to whether or not the answers to survival questions have predictive value for real longevity (Viscusi and Hakes, 2003). Moreover, sceptics point at a heaping of responses at focal-point values of 0, 50 or 100 percent, which hints at biased response (Bruine de Bruin *et al.*, 2002). On the other hand, an individual has access to superior information about herself than is incorporated in a life table. For a discussion, see e.g. Peracchi & Perotti (2011) or Wübker (2012b). Peracchi & Perotti (2011) using SHARE data and Smith et al. (2001) using HRS data find evidence that subjective beliefs about longevity relate to observed survival patterns. For our purpose, however, it is not crucial whether or not individual beliefs are an accurate reproduction of reality, since the prevention decisions of individuals will be influenced by their subjective beliefs including biases.

There are a limited number of variables that relate to risk of a specific disorder. In the empirical analysis of breast cancer prevention, we take up an indicator for whether or not the individual has had a positive cancer diagnosis (except breast cancer) in the past. We believe that the experience of another cancer will increase the subjective belief (and/or objective risk) of developing breast cancer. The model for dental prevention is enlarged with a variable indicating whether or not the individual experiences biting problems and has dentures.

Control variables used in the empirical model are age (in classes of 5 years); education (based on ISCED-97 scale)[20]; dummies for gender, partner, house owner, nationality (native, EU-citizen or non-EU citizen), (past) smoker; and country dummies by wave. These control variables capture elements of awareness, prevalence, need, future utility, subjective beliefs and risk aversion.

Macro data Country dummies capture the effect of intercountry variation, but remain a blackbox with respect to the underlying causes. While they are necessary when comparing countries (and are also present in our model), we enrich the SHARE data with information about health policies and health indicators from other sources. These can be seen as rough and partial measures of α and c_α. The additional data are not individual specific but group or region specific. Due to missing data or lack of comparable information on health policies, Israel and Switzerland are left out of the analysis and only data from the 13 remaining countries are used (Austria, Belgium, Czech Republic, Denmark, France, Germany, Greece, Ireland, Italy, the Netherlands, Poland, Spain, Sweden).

For breast cancer, the WHO GLOBOCAN project provides age and country specific information on incidence and mortality rates for 2008. The rates are expressed in cases per 1000 individuals. The report on cancer screening in the European Union provides information on the type of screening program (population-based or opportunistic[21]) and

[20] We create dummies for highest educational degree: primary education (ISCED 0-1), lower secondary education (ISCED 2), upper secondary education (ISCED 3-4), higher vocational education and university degree (ISCED 5-6).

[21] By population-based screening, we refer to an organized screening program (with a specified target group, a specific screening test, intervals, quality assurance, monitoring and other procedures) managed by an organization at a national or regional level. In addition to the high degree of organization, every eligible individual served by the screening program is individually identified and personally invited to attend screening. Opportunistic screening on the other hand refers to screening outside an organised program and without personal invitation. The initiative to perform a screening examination is taken either by the individual or the health-care provider. Opportunistic screening may or may not be performed according to the public screening policy (if one exists), e.g. it may be applied to individuals outside the targeted population or according to a different screening technique.

the implementation status[22] (von Karsa et al., 2008). In Germany, Denmark and Italy, population based programs are administered at a regional level, with varying progress in program implementation. We include the region-specific information on the implementation status of the breast cancer screening program in our dataset. Moreover, von Karsa et al. provide details on the country target group for screening and on the chances of receiving an invitation letter per country. All of this information was matched with the characteristics of the individuals in our sample. For Spain and Sweden, regional differences in target group definitions were taken into account.

Information on influenza vaccination policies and country differences can be found on the website and in the publications from the VENICE project.[23] Many countries define different target groups for influenza vaccination based on age (e.g. individuals aged 65 and over), on existing illnesses (e.g. individuals with chronic lung diseases) and on professions that have interactions with vulnerable groups (e.g. health care workers). We replicated the target groups based on the specific rules for each country. In addition, we can distinguish between three reimbursement schemes: free vaccination, partially-subsidized vaccination, or no subsidies.

Comparable regional or country information for the other disorders is limited or inaccurate, and is not included.

1.4.3 Results

Table 1.5 presents the averaged individual marginal effects of the participation determinants in the prevention of the different disorders. The analysis is performed on a large number of individuals: 11,350 individuals for breast cancer screening, 35805 individuals for dental prevention of whom 12972 have entries in both wave 1 and 2, 21495 individuals for influenza vaccination, 4029 individuals for cholesterol screening, 3339 for blood pressure screening, and, 5649 for blood sugar screening. The analysis confirms results previously obtained with SHARE by Schmitz and Wübker (2011), Jusot et al. (2012) and Wübker (2012a, 2012b) for breast cancer screening and influenza vaccination, but some explanatory and control variables differ to match better our theoretical model. Of course, since all our data are basically taken from a cross-section (and it is not possible to find convincing instruments), it would be wrong to give a causal interpretation to our results.

[22] It takes time to set up a population-based program. By implementation status, we refer to the progress made in this process. The starting point is a planning phase, followed by a pilot project, a rollout over the entire region/country and finally a completed population-based screening program.

[23] VENICE is an acronym for Vaccine European New Integrated Collaboration Effort.

Significant effects should be interpreted as associations. SHARE targets individuals aged 50 years and over (plus spouses), which implies that individuals below 50 years old are by definition partners and might not constitute a representative sample of the population. We keep all individuals in our sample regardless of age, but perform the empirical analysis also on individuals aged 50 and over (see Table 1.8 in Appendix 5). The empiricial results and conclusions are similar.

The results in Table 1.5 show that a decrease in background health status, represented by a higher BMI, ADL index or mobility index and a lower score on grip strength are either insignificant or have the hypothesized sign. The pattern that emerges from the self-assessed health dummies also confirms the hypothesis that a decrease in background health decreases participation in breast cancer screening and dental prevention and increases participation in prevention for the other disorders. Even though our results are to be interpreted cautiously, it is very reassuring that the inclusion of multiple health variables, capturing divergent aspects of an individual background health, lead to partial effects that go in the same direction and that confirm the hypothesized correlations.

The marginal effects of self-assessed health are most pronounced for the types of prevention that can be provided by a general practitioner (GP), i.e. influenza vaccination, cholesterol screening, blood pressure screening and blood sugar screening. An alternative explanation for these results could be that individuals with worse background health simply visit more often a GP, who might inform them about preventive actions. In this way, participation in prevention can be carried out in combination with a visit for another reason and individuals can be encouraged to take-up prevention in ways not directly captured in our model. We test whether this alternative channel is driving our results in two ways. First, we perform the baseline analysis on the subsample of individuals who have visited a GP in the past 12 months (results available on request). The effects for breast cancer screening, dental prevention and influenza vaccination are almost identical to the baseline results. With respect to the different blood tests, the significant effects among the objective indicators remain present and indicate that a decreasing health leads to a higher take-up of the test. The marginal effects of self-assessed health decrease in absolute value but the pattern remains visible and significant at a lower significance level.

Table 1.5: Determinants in the take-up of prevention: health, mortality risk and income

	breast cancer screening		**dental prevention**		**influenza vaccination**		**cholesterol screening**		**blood pressure screening**		**blood sugar screening**	
Variables	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)
Self-assessed health (Ref. = very good/excellent)												
SAH: good	0.012	(0.010)	-0.008	(0.005)	0.038	(0.007)***	0.081	(0.019)***	0.080	(0.019)***	0.081	(0.017)***
SAH: fair	0.007	(0.013)	-0.023	(0.006)***	0.072	(0.009)***	0.099	(0.024)***	0.123	(0.026)***	0.102	(0.020)***
SAH: poor	-0.022	(0.020)	-0.024	(0.010)**	0.089	(0.014)***	0.133	(0.036)***	0.151	(0.041)***	0.158	(0.029)***
Objective health indicators												
ADL index	-0.072	(0.043)*	-0.040	(0.024)*	0.008	(0.030)	0.059	(0.090)	-0.034	(0.114)	0.131	(0.069)*
Mobility index	0.013	(0.021)	-0.026	(0.012)**	0.046	(0.016)***	0.051	(0.046)	0.200	(0.056)***	0.067	(0.036)*
BMI	-0.002	(0.001)*	-0.004	(0.000)***	0.002	(0.001)***	0.008	(0.002)***	0.001	(0.002)	0.006	(0.002)***
Grip strength dominant hand	0.000	(0.001)	0.000	(0.000)*	-0.001	(0.000)***	0.000	(0.001)	0.000	(0.001)	0.001	(0.001)
Importance of the future												
Prob. death in 10 years	-0.062	(0.026)**	-0.017	(0.012)	-0.011	(0.016)	-0.012	(0.057)	0.084	(0.070)	-0.049	(0.044)
Income (Ref. = decile 1)												
Decile 2	-0.022	(0.019)	-0.006	(0.011)	-0.032	(0.016)**	-0.016	(0.032)	0.005	(0.033)	-0.036	(0.028)
Decile 3	-0.017	(0.019)	-0.017	(0.011)	-0.001	(0.015)	-0.023	(0.032)	-0.016	(0.033)	-0.008	(0.028)
Decile 4	0.004	(0.019)	-0.003	(0.010)	-0.000	(0.015)	0.058	(0.034)*	0.013	(0.035)	0.053	(0.030)*
Decile 5	0.004	(0.019)	0.001	(0.010)	0.002	(0.015)	0.047	(0.037)	0.002	(0.037)	0.067	(0.033)**
Decile 6	0.047	(0.020)**	0.005	(0.010)	0.001	(0.015)	0.028	(0.039)	-0.007	(0.038)	0.023	(0.034)
Decile 7	0.037	(0.020)*	0.041	(0.010)***	0.023	(0.015)	0.023	(0.039)	0.015	(0.039)	0.027	(0.034)
Decile 8	0.049	(0.020)**	0.041	(0.010)***	0.019	(0.015)	0.049	(0.038)	0.012	(0.037)	0.056	(0.033)*
Decile 9	0.044	(0.020)**	0.053	(0.010)***	0.024	(0.015)*	0.059	(0.038)	0.038	(0.037)	0.046	(0.033)
Decile 10	0.058	(0.020)***	0.053	(0.010)***	0.024	(0.015)*	0.057	(0.039)	0.051	(0.038)	0.057	(0.034)*
Breast cancer specific												
Diagnosed cancer (except breasts)	0.085	(0.025)***										
Dental specific												
Dentures			-0.118	(0.005)***								
Trouble biting			-0.049	(0.005)***								
No. of observations	11350		48777		21495		4029		3339		5649	

Note: Averaged marginal effects from probit regressions are reported with robust standard errors. For dental prevention, standard errors are clustered at the individual level. Significance levels of coefficients: * p<0.10, ** p<0.05, All regressions control for education, nationality, gender, age, partner, past an current smoker, house owner, country dummies by wave as discussed in section 1.4.2.
Database: SHARE, wave 1 and wave 2

Second, we introduce two additional explanatory variables to our baseline specification, i.e. the number of visits to the GP in the past 12 months and a GP quality index. We adopt the GP quality index as proposed by Wübker (2012b). The GP quality index is not available for all individuals. Using it reduces the number of observations by more than 60% for dental prevention, by 22% for breast cancer screening and influenza vaccination and by around 8% for the other disorders. The results can be found in Table 1.9 in Appendix 5. The inclusion of GP visits and GP quality reduces the point estimates of the marginal effects as well as their significance levels. Overall, the hypothesized correlations remain present, both for objective health indicators and for self-assessed health. Moreover, the results show that individuals who visited a GP more frequently in the past year are significantly more likely to participate in all types of prevention and that the quality of the GP has a positive effect on all types of prevention except dental prevention. These results stand to reason. Introducing the GP-variables does not alter considerably the overall pattern for the other variables (the most pronounced change is a decrease of the income effect for dental prevention).

Also as expected, mortality risk over 10 years is an important predictor in the model of breast cancer screening, but has no significant effect on any other type of prevention. Sensitivity analysis confirms these results. The signs and significance do not change when we vary the death rate gradually from $k = 4$ to $k = 8.8$. For k between 8.8 and 10, the results are borderline significant with p-values between 10% and 15%. Only in the model of blood sugar tests, an increase in mortality risk over 10 years with $4 \leq k < 4.6$, slightly decreases the probability of participation at a significance level of 10%. However, since SHARE is oriented towards individuals aged 50 and over, higher values for k, i.e. a more important increase in the probability of dying as one ages, are more probable.

Controlling for education levels, we find a positive effect of income on preventive behavior, although not always significant. We find that in particular deciles 1 to 3/4 participate less in prevention. The income effects are most pronounced for breast cancer screening and dental prevention. The former is in line with the theoretical predictions. An alternative income specification, i.e. defining income deciles at the country level (by wave), does not change our results in any important way (results available upon request).

Next, we have individual specific information that serves as proxy for (subjective and/or objective) disease risk. We find that an earlier diagnosis of non-breast cancer increases the probability of participation in breast cancer screening on average by 8.5 percentage points. This can be explained by higher (subjective and/or objective) risk of developing breast

Table 1.6: Determinants in the take-up of prevention: macro data on policy design

	breast cancer screening		**influenza vaccination**	
Variables	Marginal effects (SE)		Marginal effects (SE)	
Breast cancer specific				
Age and country specific incidence	-0.007	(0.018)		
Age and country specific mortality	0.141	(0.058)**		
Country target group	0.060	(0.024)**		
Prob. receiving invitation letter	0.172	(0.023)***		
Pop. based program complete	0.179	(0.021)***		
Influenza specific				
Country target group based on age			0.079	(0.017)***
Country target group based on illness			0.058	(0.008)***
Subsidized vaccination			0.085	(0.014)***
Free vaccination			0.162	(0.014)***

Note: Coefficient estimates from probit regressions are reported with robust standard errors. For dental prevention, standard errors are clustered at the individual level. Significance levels of coefficients: * p<0.10, ** p<0.05, All regressions control for health, mortality risk, income, education, nationality, gender, age, partner, past an current smoker, house owner, country dummies by wave as discussed in section 1.4.2. In addition, the regressions for breast cancer screening includes a dummy for having had a cancer (except breast cancer)
Database: SHARE, wave 1 and wave 2

cancer or by an increased attention on the part of the health care providers. Preventive dental care is negatively related to having dentures and having trouble biting. This is not surprising, since the former probably reduces the need for regular preventive care, while the latter requires curative rather than preventive care.

In Table 1.6, we add the additional information from macrosources to the microdata from SHARE. With the exception of breast cancer incidence, all variables show very significant positive marginal effects. We observe that the participation decision is positively affected by age and country-specific mortality rates. Breast cancer screening is higher for individuals who are targeted, who receive an invitation letter and who live in a country or region that has fully enacted a population based program. The probability of taking up a vaccine increases if an individual belongs to a target group. Finally, we observe that monetary stimuli clearly increase the probability of receiving influenza vaccination, by 9 percentage points for subsidized vaccination and an as could be expected an even stronger effect of 16 percentage points for free vaccinations. We conclude that policy features play a very important role in the take-up of prevention. They do this without significantly altering the marginal effects of health, income and mortality risk, they rather explain

some of the intercountry variation.

1.5 Conclusion

We analyzed participation in medical prevention with an expected utility model. Rather than focusing on one specific intervention, we aimed to explain the differences for various prevention procedures within one coherent model. This model is sufficiently flexible to distinguish primary and secondary prevention for either fatal or non-fatal diseases. Moreover, we integrated the idea of different disease types characterized by a different interaction with background health. The model yields different predictions in the different cases. We tested these predictions with individual data from SHARE and the model performed reasonably well.

Our main contribution is the construction of a flexible theoretical model. We believe that this is useful. Starting from a rather general model allows us to bring some coherency into the disparate insights from the empirical literature, and to validate differentiated hypotheses for different cases.[24] In this respect, the expected utility model (broadly interpreted) seems to be an interesting starting point for further developments. These developments should go in two directions.

First, our theoretical model can be refined further. Introducing a richer dynamic structure would make it possible to integrate past behavior and more sophisticated expectations into the explanatory framework.[25] More fundamentally, there are by now sufficient indications in the literature that the expected utility model cannot explain all of the empirical regularities, not even when it is interpreted – as in our model – in a purely subjective way, taking due account of biases in the perception of costs and probabilities. Recent papers in the behavioral economic literature have built in other realistic features into the analysis of screening and prevention decisions: hyperbolic discounting and myopia (Byrne and Thompson, 2001; Fang and Wang, 2010), loss-aversion over changes in beliefs (Fels, 2011), biased perceptions of risks in a rank-dependent utility model (Etner and Jeleva, 2013) and anticipatory feelings (Oster et al., 2011). While some of these developments are very promising, it would be overly ambitious to try to build a general model of different prevention decisions incorporating these mechanisms. For a comparative exercise, the expected utility model remains a convenient and flexible starting point. However, it should

[24] A similar position is taken by Howard (2005, p. 893).

[25] Some authors have used information in the third wave of SHARE to analyse the influence of reported past behavior in regard to (non-)participation in breast cancer screening (Wübker, 2012a, 2012b) and in preventive dental care (Listl et al., 2012).

be checked how much the more sophisticated behavioral models add to the explanatory power of an (extended) expected-utility model, especially in cases of primary prevention and secondary prevention with screening as a necessary condition for treatment.[26]

Second, on the empirical side, we lack data on important parameters such as the subjective rate of time preference or the subjective perception of probabilities. Future work should try to collect direct measures of these parameters.[27] Using such well-designed measures would allow a more convincing testing of the hypotheses.

Throughout the chapter, we have focused on medical prevention procedures, mainly because participation is modeled in a binary way, and we analyzed the prevention of a particular disorder, rather than an improvement of the general health status. Our model can be extended to include lifestyle choices, such as physical activity, eating healthy or (quitting) smoking, as long as the preventive decision can be defined in a binary way and the disorders that are prevented can be well-delineated.[28] The extensions in this context could include elements of ability (e.g. the ability to sport), preferences (for certain types of food, activities) and addiction (for alcohol, smoking, etc.).

[26] The strongest arguments against using the expected utility model can be found in Oster et al. (2011). However, they analyse medical testing decisions for Huntington's disease – where at this moment no curative treatment is available.

[27] Examples in the literature are Bradford et al. (2010) for time preferences and Carman and Kooreman (2011) for subjective probabilities.

[28] Take physical exercise as an example. First, we can define half an hour of physical activity per week as the threshold of participation in prevention. Above (below) the threshold, individuals (do not) participate in prevention. However, there are many other potential thresholds. Second, engaging in regular physical activity contributes to the prevention of - amongst others - cardiovascular diseases, obesity and depression. Note that it might be difficult to define the disease characteristics when several diseases are prevented at the same time. It is, however, always possible to focus on the main prevented disease or group diseases with similar characteristics.

Appendix 1: Relaxing the separability assumption

Empirically, there is no consensus upon the sign of the interaction between health and income in the utility function. We therefore assumed separability throughout the main text. In this Appendix we explore alternative assumptions. A positive relationship implies $u_{12}(y,h,m) > 0$, i.e. an individual enjoys an additional unit of income more when she has a better general background health, or vice versa, when the individual earns a higher income, she values health more. A negative relationship is defined as $u_{12}(y,h,m) < 0$, which means that an individual enjoys an additional unit of income more when she has a worse general background health, or vice versa, when the individual has a lower income, she values health more.

The general comparative static results for income are to be compared with eqs. (1.32) and (1.32'). For secondary prevention this gives:

$$\begin{aligned}\frac{\partial \Delta EU_1}{\partial y_1} &= p_1 \times se \times u_1(y_1 - c_\alpha - c_e, h_1, e) + (1-p_1) \times [u_1(y_1 - c_\alpha, h_1, 0) - u_1(y_1, h_1, 0)] \quad (1.35)\\ &+I(nf)\left[\begin{array}{c} p_1 \times (u_1(y_1 - c_\alpha - c_l, h_1, l) - u_1(y_1 - c_l, h_1, l)) \\ -p_1 \times se \times u_1(y_1 - c_\alpha - c_l, h_1, l) \end{array}\right]\end{aligned}$$

and for primary prevention:

$$\begin{aligned}\frac{\partial \Delta EU_1}{\partial y_1} &= p_1 \times se \times u_1(y_1 - c_\alpha, h_1, 0) + (1-p_1) \times [u_1(y_1 - c_\alpha, h_1, 0) - u_1(y_1, h_1, 0)] \quad (1.35')\\ &+I(nf)\left[\begin{array}{c} p_1 \times (u_1(y_1 - c_\alpha - c_l, h_1, l) - u_1(y_1 - c_l, h_1, l)) \\ -p_1 \times se \times u_1(y_1 - c_\alpha - c_l, h_1, l) \end{array}\right]\end{aligned}$$

For a fatal disease, only the first line matters, which is always positive, no matter how the relationship between income and health is specified. For a non-fatal disease, the overall effect is unknown, since the second line can be either positive or negative. However, the marginal effect can be ranked according to the relationship between y and m. The overall partial effect will be *ceteris paribus* higher the more positive the relationship between y and m.

The general comparative static results for health are to be compared with eqs. (1.33) and (1.33'). For secondary prevention this gives:

$$\begin{aligned}\frac{\partial \Delta EU_1}{\partial h_1} &= p_1 \times se \times u_2(y_1 - c_\alpha - c_e, h_1, e) + (1-p_1) \times [u_2(y_1 - c_\alpha, h_1, 0) - u_2(y_1, h_1, 0)] \quad (1.36)\\ &+I(nf)\left[\begin{array}{c} p_1 \times (u_2(y_1 - c_\alpha - c_l, h_1, l) - u_2(y_1 - c_l, h_1, l)) \\ -p_1 \times se \times u_2(y_1 - c_\alpha - c_l, h_1, l) \end{array}\right]\end{aligned}$$

and for primary care:

$$\begin{aligned}\frac{\partial \Delta EU_1}{\partial h_1} &= p_1 \times se \times u_2(y_1 - c_\alpha, h_1, 0) + (1 - p_1) \times [u_2(y_1 - c_\alpha, h_1, 0) - u_2(y_1, h_1, 0)] \quad (1.36') \\ &+ I(nf) \left[\begin{array}{c} p_1 \times (u_2(y_1 - c_\alpha - c_l, h_1, l) - u_2(y_1 - c_l, h_1, l)) \\ -p_1 \times se \times u_2(y_1 - c_\alpha - c_l, h_1, l) \end{array} \right]\end{aligned}$$

The partial effects for health are more complex. For fatal diseases, the partial effect is positive when health and income are independent or negatively correlated. The effect is unknown when health and income are positively correlated. For non-fatal diseases, we are only able to predict the sign of the marginal effect if income and health are independent. The results in this case are the same as for eqs. (1.33) and (1.33'). When income and health are negatively or positively correlated, the overall effect is unknown, since both positive and negative terms are present and their relative weight (given by p_1 and se) determines the overall sign.

Appendix 2: The T period model

Our simplified two period model can be generalized to a multi-period model. In our approach, decisions in the different time periods are independent of past decisions. See, e.g. de la Mata (2011) and Etner and Jeleva (2013) for a richer dynamic specification. Take the number of periods to be T; T can be individually specific. We assume that in period T the individual dies, so that $V_T = 0$. We solve the problem backwards. In this Appendix, we keep the non-separable utility function. We introduce an indicator $I(prim)$ to indicate primary prevention. This makes it easier to incorporate primary and secondary prevention within one specification.

We can define ΔEU_{T-1} to incorporate both primary and secondary prevention:

$$\begin{aligned}\Delta EU_{T-1} &= p_{T-1} \times se \times [(1 - I(prim))u(y_{T-1} - c_\alpha - c_e, h_{T-1}, e) + I(prim)u(y_{T-1} - c_\alpha, h_{T-1}, 0)] \\ &+ (1 - p_{T-1}) \times u(y_{T-1} - c_\alpha, h_{T-1}, 0) \\ &+ I\,(nf) \times p_{T-1}(1 - se)u(y_{T-1} - c_\alpha - c_l, h_{T-1}, l) - f(\alpha) \\ &- (1 - p_{T-1}) \times u(y_{T-1}, h_{T-1}, 0) - I\,(nf) \times p_{T-1}u\,(y_{T-1} - c_l, h_{T-1}, l) \\ &+ (1 - I(nf)) \times p_{T-1} \times se \times \beta(1 - p_{x,T})V_T \quad (1.37) \\ &= \Delta CPEU_{T-1} \\ &+ (1 - I(nf)) \times p_{T-1} \times se \times \beta(1 - p_{x,T})V_T \quad (1.38)\end{aligned}$$

We can subdivide ΔEU_{T-1} into two terms: a first term captures the current period difference in expected utility ($\Delta CPEU_{T-1}$) and a second term represents future utility. We can also define V_{T-1}, which captures expected utility from period $T-1$ onwards. Since expected utility depends on preventive behavior, we introduce an indicator function $I_{T-1}(part)$ that equals 1 if the individual participates in prevention in period $T-1$ (i.e. $\Delta EU_{T-1} > 0$) and zero if the individual does not take part in prevention (i.e. $\Delta EU_{T-1} < 0$).

$$\begin{aligned} V_{T-1} &= (1-p_{T-1}) \times u(y_{T-1}, h_{T-1}, 0) + p_{T-1} I(nf)\, u(y_{T-1} - c_l, h_{T-1}, l) \\ &\quad + I_{T-1}(part) \Delta EU_{T-1} \\ &= (1-p_{T-1}) \times u(y_{T-1}, h_{T-1}, 0) + p_{T-1} I(nf)\, u(y_{T-1} - c_l, h_{T-1}, l) \\ &\quad + I_{T-1}(part) \Delta CPEU_{T-1} + I_{T-1}(part) \times p_{T-1} \times se \times (1 - I(nf))\, \beta (1-p_{x,T}) V_T \end{aligned} \tag{1.39}$$

It is clear from eqs. (1.38) and (1.39), that all references to future utility disappear for a non-fatal disease ($I(nf) = 1$). Therefore, in that case the prevention decision depends only on current period variables, and the comparative static results are the same as in the two period-model.

The analysis for fatal diseases is more challenging, since in this case future utility will not disappear from the model if the individual participates in prevention. For any period t, we can characterize the decision process and the payoff as follows:

$$\begin{aligned} \Delta EU_t &= \Delta CPEU_t + p_t \times se \times \beta \times (1 - p_{x,t+1}) \times \\ &\quad \sum_{i=t+1}^{T-1} \left[I_i(part)\, \Delta CPEU_i + (1-p_i)\, u_i^{HE} \right] \times \prod_{j=t+1}^{i-1} \left[p_j \times se \times \beta\, (1 - p_{x,j+1})\, I_j(part) \right] \end{aligned} \tag{1.40}$$

with

$$\begin{aligned} \Delta CPEU_t &= p_t \times se \times [(1 - I(prim)) u(y_t - c_\alpha - c_e, h_t, e) + I(prim) u(y_t - c_\alpha, h_t, 0)] \\ &\quad + (1 - p_t) \times [u(y_t - c_\alpha, h_t, 0) - u(y_t, h_t, 0)] - f(\alpha) \\ V_t &= \sum_{i=t}^{T-1} \left[I_i(part)\, \Delta CPEU_i + (1-p_i)\, u_i^{HE} \right] \times \prod_{j=t}^{i-1} \left[p_j \times se \times \beta\, (1 - p_{x,j+1})\, I_j(part) \right] \end{aligned} \tag{1.41}$$

In the case of a fatal disease, expected utility consists of current period expected utility and future utility. Future utility becomes more important when the individual expects to live longer ($T-t$ larger), when the individual is more future-oriented (β larger), when the mortality risk for other diseases is lower ($p_{x,j+1}$ smaller) and when the benefit from prevention is more important ($p_j \times se$ larger). In the expression for future utility, we take into account the individual's future prevention decisions. If prevention has a positive payoff in the future, this payoff will be taken into account for current decisions as well.

Comparative statics The comparative statics are similar to those in the two period model. Note, however, that age (or, more accurately the individual's time horizon $T-t$), becomes relevant in the generalized model. In what follows, we look at the comparative statics of fatal diseases.

With respect to the **future**, we can conclude that the partial effects have the same sign, but since V_t gets smaller as an individual ages, the effect of β and $p_{x,t+1}$ decreases over time.

For the **disease characteristics** and **treatment costs**, the partial effects have the same sign as in the two period model, but the time horizon and the future prevention decision will influence the magnitude of the effect. We assume for simplicity that the test characteristics and the costs of treatment are the same in each period. However, this assumption can be relaxed.

A higher **subjective probability** of having the disorder in period t still leads to an increase in participation in the same period. However, the effect of p_{t+1} on the probability of participation in period t is not the necessarily the same as in the two periods model:

$$\begin{aligned}\frac{\partial \Delta EU_t}{\partial p_{t+1}} &= p_t \times se \times \beta \times (1-p_{x,t+1}) \times \left[I_{t+1}(part) \frac{\partial \Delta CPEU_{t+1}}{\partial p_{t+1}} - u_{t+1}^{HE} \right] \\ &\quad + p_t \times se^2 \times \beta^2 \times (1-p_{x,t+1}) \times (1-p_{x,t+2}) \times I_{t+1}(part) \times \\ &\quad \sum_{i=t+2}^{T-1} \left[I_i(part) \Delta CPEU_i + (1-p_i) u_i^{HE} \right] \times \prod_{j=t+2}^{i-1} \left[p_j \times se \times \beta (1-p_{x,j+1}) I_j(part) \right]\end{aligned} \tag{1.42}$$

We can distinguish between two terms. On the one hand there is a direct effect in period $t+1$, which is negative. If p_{t+1} increases, the individual is more likely to die, and utility decreases. This decrease cannot be countered by the direct positive effect of prevention in period $t+1$. On the other hand, there might be an indirect effect of prevention in the subsequent periods. If the individual participates in prevention in period $t+1$ ($I_{t+1}(part)=1$), she reduces the risk from dying and gains utility in periods $t+2, t+3, ..., T-1$. The total utility gained depends, amongst other factors, on the participation decision in the subsequent periods and the time horizon. The second term can have a positive indirect effect on the participation decision in period t. The overall effect is ambiguous.

The partial effect of current **income** does not change. The effect of a future marginal change in income y_j with $t < j < T$ still has a positive effect on participation in the current period. Finally, the partial effect of both current and future **health** in case of a fatal disease is always positive, as long as health between the periods is independent or positively correlated.

Appendix 3: First order Taylor expansion

We start from eq. (1.32) and perform a Taylor expansion around y_1, for secondary prevention, this gives:

$$\begin{aligned}\frac{\partial \Delta EU_1}{\partial y_1} &= v_1(y_1) \times p_1 \times se - v_{11}(y_1)\left[c_\alpha \times (1 - p_1 \times (1 - se)) + c_e \times p_1 \times se\right] \\ &\quad -I(nf)\left[v_1(y_1) \times p_1 \times se - v_{11}(y_1) \times \left[c_\alpha \times (p_1 \times (1 - se)) + c_l \times p_1 \times se\right]\right]\end{aligned} \tag{1.43}$$

and for primary prevention:

$$\begin{aligned}\frac{\partial \Delta EU_1}{\partial y_1} &= v_1(y_1) \times p_1 \times se - v_{11}(y_1)\left[c_\alpha \times (1 - p_1 \times (1 - se))\right] \\ &\quad -I(nf)\left[v_1(y_1) \times p_1 \times se - v_{11}(y_1) \times \left[c_\alpha \times (p_1 \times (1 - se)) + c_l \times p_1 \times se\right]\right]\end{aligned} \tag{1.44}$$

Since $v_1(y_1) > 0$ and $v_{11}(y_1) \leqslant 0$, the effect will always be positive for a fatal disease. For a non-fatal disease, we derive the following Taylor condition for secondary prevention:

$$\frac{\partial \Delta EU_1}{\partial y_1} = -v_{11}(y_1) \times \left[c_\alpha + c_e \times p_1 \times se - c_l \times p_1 \times se\right] \tag{1.45}$$

$$\frac{\partial \Delta EU_1}{\partial y_1} \geqslant 0 \Leftrightarrow c_\alpha - p_1 \times se \times (c_l - c_e) \geqslant 0 \Leftrightarrow c_\alpha \geqslant p_1 \times se \times (c_l - c_e) \tag{1.46}$$

and for primary prevention:

$$\frac{\partial \Delta EU_1}{\partial y_1} = -v_{11}(y_1)\left[c_\alpha - c_l \times p_1 \times se\right] \tag{1.47}$$

$$\frac{\partial \Delta EU_1}{\partial y_1} \geqslant 0 \Leftrightarrow c_\alpha - c_l \times p_1 \times se \geqslant 0 \Leftrightarrow c_\alpha \geqslant p_1 \times se \times c_l \tag{1.48}$$

Appendix 4: Comparative static results

Characteristics of the testing procedure Starting from eq. (1.18), we derive for secondary prevention:

$$\frac{\partial \Delta EU_1}{\partial se} = p_1(u^P - u^{FN}) > 0 \tag{1.49}$$

where the conclusion about the sign follows from eq. (1.20). An improvement of the effectiveness of prevention, without additional monetary or psychological costs, always makes prevention more attractive. For primary prevention, we have

$$\frac{\partial \Delta EU_1}{\partial se} = p_1(u^{TN} - u^{FN}) > 0 \text{ for } ef = se \tag{1.50}$$

The comparative static results are straightforward for the "cost" parameters α and c_α. We have $\frac{\partial u^{xx}}{\partial z} < 0$, for $z = (\alpha, c_\alpha)$ and for $xx = (P, TN, FN)$. We therefore conclude that

$$\frac{\partial \Delta EU_1}{\partial \alpha} < 0 \tag{1.51}$$

$$\frac{\partial \Delta EU_1}{\partial c_\alpha} < 0 \tag{1.52}$$

As could be expected, increased costs make preventive effort less attractive. If an increase in α leads to an increase in c_α, the negative effects are reinforced. If, on the other hand, a policy change increases α, and, at the same time, se, positive and negative effects should be weighed against each other.

Characteristics of the disease The effect of a change in p_1 is less straightforward. Taking the derivative of eq. (1.17), we get for secondary prevention:

$$\frac{\partial \Delta EU_1}{\partial p_1} = \left[u^{HE} - u^{TN}\right] + \left[se \times u^P + (1-se)u^{FN} - u^S\right] \tag{1.53}$$

$$= [v(y_1) - v(y_1 - c_\alpha)] + se \times [v(y_1 - c_\alpha - c_e) + w(h_1, e)) + \beta(1 - p_{x,2})V_2] \tag{1.54}$$

$$+I(nf) \times [v(y_1 - c_\alpha - c_l) - v(y_1 - c_l) - se \times (v(y_1 - c_\alpha - c_l) + w(h_1, l) + \beta(1 - p_{x,2})V_2)]$$

which has an obvious interpretation. The relative ranking of utility states in eq. (1.20) shows clearly that if the individual is healthy (states u^{HE}, u^{TN}), participation in prevention leads to additional costs and a utility loss, while if she is ill (states u^S, u^P, u^{FN}), it depends on the underlying parameters, such as the costs and the efficiency of the preventive procedures, whether prevention leads to a gain or a loss. As p_1 increases there is a shift away from the utility loss when healthy, towards the utility gain or loss when sick. The former leads to a positive effect on participation in prevention, captured by the first term in eq. (1.53), while the latter may result in a positive or a negative effect on preventive behavior, captured by the second term in eq. (1.53). The positive effect will dominate, i.e. $\frac{\partial \Delta EU_1}{\partial p_1} > 0$, for a fatal disease and for preventive procedures with a high sensitivity se and/or low screening costs c_α. For primary prevention, the partial effect is similar, but u^P is replaced by u^{TN}, which *ceteris paribus* leads to a higher marginal effect:

$$\frac{\partial \Delta EU_1}{\partial p_1} = \left[u^{HE} - u^{TN}\right] + \left[se \times u^{TN} + (1-se)u^{FN} - u^S\right] \text{ for } ef = se \tag{1.55}$$

$$= [v(y_1) - v(y_1 - c_\alpha)] + se \times [v(y_1 - c_\alpha) + w(h_1, 0) + \beta(1 - p_{x,2})V_2] \tag{1.56}$$

$$+I(nf) \times [v(y_1 - c_\alpha - c_l) - v(y_1 - c_l) - se \times (v(y_1 - c_\alpha - c_l) + w(h_1, l) + \beta(1 - p_{x,2})V_2)]$$

We can also draw conclusions about the effect of p_2 on the probability of taking a

preventive test in period 1. As noted before, it will only have an impact for fatal diseases. In that case, we get from eqs. (1.27) and (1.31) that

$$\frac{\partial \Delta EU_1}{\partial p_2} = -p_1 \times se \times \beta(1 - p_{x,2}) \left(v(y_2) + w(h_2, 0)\right) < 0 \tag{1.57}$$

The intuition is obvious. Future utility V_2 unambiguously decreases as p_2 increases, since the individual is less likely to be healthy and more likely to be dead. As a result ΔEU_1 decreases and prevention becomes less interesting. This is in accordance with the conclusions from eq. (1.31).[29]

A last characteristic of the disease is the treatment cost, represented in the model by c_e and c_l. Starting from eqs. (1.28) and (1.28'), we get:

$$\frac{\partial \Delta EU_1}{\partial c_e} = -p_1 \times se \times v_1(y_1 - c_\alpha - c_e) \leqslant 0 \tag{1.58}$$

$$\frac{\partial \Delta EU_1}{\partial c_l} = I(nf) \times p_1 \times [v_1(y_1 - c_l) - (1 - se) \times v_1(y_1 - c_\alpha - c_l)] \tag{1.59}$$

An increase in the cost of early treatment only matters for secondary prevention. It leads to a reduction in ΔEU_1 and, consequently, lowers the incentives for preventive action. Higher curative (late stage) treatment costs have no effect for fatal diseases, since no cure is available. For non-fatal diseases the effect is ambiguous, since the costs can occur both in case of participation (state u^{FN}) as in case of non-participation (state u^S). However, if se is high enough and/or c_α low, more expensive curative treatment increases the incentives for preventive effort. That was only to be expected. Prevention is the only possibility to avoid the larger cost, but this cost avoidance can only work if prevention is reasonably effective (se high enough) and screening costs are limited.

Appendix 5: Additional empirical results

[29] If the time horizon is longer, as in the multi-period model, the effects of p_2 become more complex (see Appendix 2). Given that the individual can choose to participate in prevention in period 2 as well, she can counter partly the utility loss due to an increased risk of illness.

Table 1.7: Descriptive statistics

	Min	Max	Mean	Standard deviation
Breast cancer screening	0	1	0.540	(0.498)
Dental prevention	0	1	0.405	(0.491)
Influenza vaccination	0	1	0.327	(0.469)
Cholesterol screening	0	1	0.532	(0.499)
Blood pressure screening	0	1	0.694	(0.461)
Blood sugar screening	0	1	0.537	(0.499)
Number of GP visits	0	98	4.586	(7.173)
GP quality index	0	1	0.253	(0.234)
SAH: very good or excellent	0	1	0.290	(0.454)
SAH: good	0	1	0.381	(0.486)
SAH: fair	0	1	0.239	(0.427)
SAH: poor	0	1	0.090	(0.286)
ADL index	0	1	0.036	(0.134)
Mobility index	0	1	0.175	(0.268)
BMI	11	78	26.529	(4.436)
Grip strength dominant hand (average of 2 attempts)	0	91	29.551	(15.006)
Prob. death in ten years	5.14e-08	1	0.122	(0.211)
Age below 40	0	1	0.002	(0.044)
Age between 40 and 44	0	1	0.007	(0.082)
Age between 45 and 49	0	1	0.026	(0.158)
Age between 50 and 54	0	1	0.168	(0.374)
Age between 55 and 59	0	1	0.185	(0.388)
Age between 60 and 64	0	1	0.166	(0.372)
Age between 65 and 69	0	1	0.145	(0.352)
Age between 70 and 74	0	1	0.120	(0.325)
Age between 75 and 79	0	1	0.091	(0.287)
Age between 80 and 84	0	1	0.058	(0.234)
Age 85 and over	0	1	0.033	(0.180)
Gender: male	0	1	0.443	(0.497)
Gender: female	0	1	0.557	(0.497)
Nationality: native	0	1	0.980	(0.140)
Nationality: EU citizen (not native)	0	1	0.012	(0.111)
Nationality: outside EU	0	1	0.007	(0.086)
Has partner	0	1	0.748	(0.434)
Schooling: primary	0	1	0.325	(0.468)
Schooling: lower secondary	0	1	0.179	(0.384)
Schooling: upper secondary	0	1	0.305	(0.460)
Schooling: university	0	1	0.191	(0.393)
House owner	0	1	0.730	(0.444)
Smoked in the past	0	1	0.472	(0.499)
Smokes currently	0	1	0.197	(0.398)
Diagnosed cancer (except breasts)	0	1	0.039	(0.195)
Breast cancer: incidence (per 1000 cases)	0.077	5	2.794	(0.883)
Breast cancer: mortality (per 1000 cases)	0.012	2	0.777	(0.463)
Breast cancer: target group	0	1	0.724	(0.447)
Breast cancer: prob. receiving invitation letter	0	1	0.381	(0.444)
Breast cancer: pop. based program complete	0	1	0.542	(0.498)
Influenza: target group (age)	0	1	0.528	(0.499)
Influenza: target group (illness)	0	1	0.259	(0.438)
Influenza: subsidized vaccination	0	1	0.334	(0.472)
Influenza: free vaccination	0	1	0.292	(0.455)

Database: SHARE, wave 1 and wave 2

Table 1.8: Determinants in the take-up of prevention: sample restricted to individuals aged 50 years and over.

	breast cancer screening		**dental prevention**		**influenza vaccination**		**cholesterol screening**		**blood pressure screening**		**blood sugar screening**	
Variables	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)
Self-assessed health (Ref. = very good/excellent)												
SAH: good	0.005	(0.011)	-0.010	(0.005)**	0.042	(0.008)***	0.080	(0.020)***	0.090	(0.019)***	0.084	(0.017)***
SAH: fair	0.003	(0.013)	-0.024	(0.006)***	0.075	(0.009)***	0.092	(0.024)***	0.127	(0.026)***	0.101	(0.021)***
SAH: poor	-0.029	(0.020)	-0.026	(0.010)***	0.095	(0.015)***	0.125	(0.037)***	0.162	(0.041)***	0.160	(0.029)***
Objective health indicators												
ADL index	-0.081	(0.043)*	-0.043	(0.024)*	0.006	(0.030)	0.052	(0.090)	-0.049	(0.113)	0.123	(0.069)*
Mobility index	0.021	(0.021)	-0.024	(0.012)**	0.047	(0.016)***	0.061	(0.047)	0.205	(0.056)***	0.075	(0.036)**
BMI	-0.001	(0.001)	-0.004	(0.001)***	0.002	(0.001)***	0.007	(0.002)***	0.000	(0.002)	0.006	(0.002)***
Grip strength dominant hand	0.001	(0.001)	0.000	(0.000)	-0.001	(0.000)***	0.000	(0.001)	0.000	(0.001)	0.001	(0.001)
Importance of the future												
Prob. death in 10 years	-0.058	(0.025)**	-0.017	(0.012)	-0.011	(0.016)	-0.012	(0.057)	0.084	(0.070)	-0.050	(0.044)
Income (Ref. = decile 1)												
Decile 2	-0.022	(0.019)	-0.003	(0.011)	-0.030	(0.016)**	-0.007	(0.033)	0.008	(0.034)	-0.031	(0.028)
Decile 3	-0.024	(0.019)	-0.017	(0.011)	0.003	(0.016)	-0.013	(0.033)	-0.003	(0.034)	-0.003	(0.029)
Decile 4	-0.002	(0.019)	-0.001	(0.010)	0.000	(0.016)	0.058	(0.035)*	0.017	(0.036)	0.054	(0.030)*
Decile 5	-0.002	(0.020)	0.002	(0.010)	0.004	(0.015)	0.060	(0.039)	0.015	(0.038)	0.074	(0.034)**
Decile 6	0.044	(0.020)**	0.007	(0.010)	0.003	(0.015)	0.036	(0.040)	-0.005	(0.039)	0.030	(0.035)
Decile 7	0.033	(0.020)	0.042	(0.010)***	0.028	(0.015)*	0.027	(0.040)	0.026	(0.040)	0.033	(0.035)
Decile 8	0.047	(0.021)**	0.041	(0.010)***	0.023	(0.015)	0.047	(0.039)	0.021	(0.038)	0.061	(0.034)*
Decile 9	0.039	(0.021)*	0.055	(0.010)***	0.027	(0.015)*	0.066	(0.039)*	0.033	(0.039)	0.053	(0.034)
Decile 10	0.045	(0.020)**	0.052	(0.010)***	0.027	(0.015)*	0.055	(0.040)	0.046	(0.039)	0.066	(0.035)*
Breast cancer specific												
Diagnosed cancer (except breasts)	0.082	(0.025)***										
Dental specific												
Dentures			-0.117	(0.005)***								
Trouble biting			-0.049	(0.006)***								
No. of observations	10614		48777		21495		4029		3339		5649	

Note: Averaged marginal effects from probit regressions are reported with robust standard errors. For dental prevention, standard errors are clustered at the individual level. Significance levels of coefficients: * p<0.10, ** p<0.05, All regressions control for education, nationality, gender, age, partner, past an current smoker, house owner, country dummies by wave as discussed in section 1.4.2.
Database: SHARE, wave 1 and wave 2

Table 1.9: Determinants in the take-up of prevention: with controls for GP visits and GP quality

Variables	breast cancer screening		dental prevention		influenza vaccination		cholesterol screening		blood pressure screening		blood sugar screening	
	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)	Marginal effects	(SE)
GP indicators												
Number of GP visits	0.002	(0.001)**	0.002	(0.001)***	0.004	(0.001)***	0.010	(0.003)***	0.029	(0.004)***	0.010	(0.002)***
GP quality index	0.063	(0.022)***	0.025	(0.015)	0.129	(0.015)***	0.476	(0.037)***	0.489	(0.043)***	0.466	(0.031)***
Self-assessed health (Ref. = very good/excellent)												
SAH: good	0.007	(0.012)	-0.007	(0.008)	0.028	(0.008)***	0.062	(0.019)***	0.048	(0.019)**	0.067	(0.017)***
SAH: fair	-0.006	(0.015)	-0.034	(0.010)***	0.051	(0.010)***	0.056	(0.026)**	0.052	(0.027)*	0.056	(0.022)***
SAH: poor	-0.021	(0.023)	-0.016	(0.017)	0.056	(0.016)***	0.059	(0.040)	0.032	(0.045)	0.071	(0.032)**
Objective health indicators												
ADL index	-0.078	(0.051)	-0.052	(0.041)	0.014	(0.036)	-0.026	(0.100)	-0.146	(0.123)	0.094	(0.077)
Mobility index	-0.007	(0.024)	-0.066	(0.019)***	0.013	(0.018)	-0.045	(0.049)	0.117	(0.059)**	0.011	(0.039)
BMI	-0.002	(0.001)**	-0.004	(0.001)***	0.001	(0.001)**	0.006	(0.002)***	-0.001	(0.002)	0.004	(0.002)***
Grip strength dominant hand	0.000	(0.001)	0.001	(0.000)*	-0.001	(0.000)*	0.001	(0.001)	0.001	(0.001)	0.002	(0.001)*
Importance of the future												
Prob. death in 10 years	-0.083	(0.031)***	-0.009	(0.022)	-0.031	(0.020)	0.014	(0.066)	0.097	(0.080)	-0.021	(0.050)
Income (Ref. = decile 1)												
Decile 2	-0.024	(0.021)	0.004	(0.017)	-0.032	(0.018)**	-0.008	(0.032)	0.004	(0.034)	-0.037	(0.028)
Decile 3	-0.014	(0.021)	-0.027	(0.017)	-0.003	(0.017)	-0.003	(0.033)	-0.009	(0.034)*	0.006	(0.029)
Decile 4	0.001	(0.021)	0.003	(0.016)	0.010	(0.017)	0.059	(0.034)*	0.021	(0.035)	0.058	(0.030)*
Decile 5	0.003	(0.022)	-0.008	(0.016)	0.009	(0.017)	0.060	(0.038)	0.006	(0.036)	0.082	(0.033)**
Decile 6	0.054	(0.023)**	-0.002	(0.017)	0.008	(0.017)	0.049	(0.039)	0.018	(0.039)	0.036	(0.034)
Decile 7	0.024	(0.023)	0.015	(0.016)	0.026	(0.017)	0.042	(0.039)	0.020	(0.038)	0.043	(0.034)
Decile 8	0.060	(0.023)***	0.029	(0.016)*	0.029	(0.017)*	0.058	(0.038)	0.016	(0.037)	0.075	(0.033)**
Decile 9	0.045	(0.023)*	0.014	(0.016)	0.030	(0.017)*	0.084	(0.038)**	0.055	(0.037)	0.074	(0.033)**
Decile 10	0.064	(0.023)***	0.020	(0.016)	0.019	(0.017)	0.074	(0.039)*	0.067	(0.037)*	0.076	(0.034)**
Breast cancer specific												
Diagnosed cancer (except breasts)	0.081	(0.028)***										
Dental specific												
Dentures			-0.113	(0.008)***								
Trouble biting			-0.026	(0.009)***								
No. of observations	8853		16874		16774		3728		3108		5164	

Note: Averaged marginal effects from probit regressions are reported with robust standard errors. For dental prevention, standard errors are clustered at the individual level. Significance levels of coefficients: * p<0.10, ** p<0.05, All regressions control for education, nationality, gender, age, partner, past an current smoker, house owner, country dummies by wave as discussed in section 1.4.2.
Database: SHARE, wave 1 and wave 2

Chapter 2

Neighborhood peer effects in the use of preventive health care

2.1 Introduction

Health and income are two major constituents of individual well-being. The first foundations for both are laid during pregnancy and childhood. A vast literature describes the impact of good nutrition and health in utero and in childhood on, amongst others, life expectancy, physical and cognitive development, schooling outcomes, labor market opportunities, and income (see e.g. Behrman, 1996; Case *et al.*, 2002; Case *et al.* 2005; Currie, 2009; Currie & Madrian, 1999; Cutler *et al.*, 2006; Van den Berg *et al.*, 2006). Children born in poor households are more likely to have worse health and begin life at a distinct disadvantage in these different domains.[1]

Poverty was widespread in Mexico around 1997. Extreme poverty is concentrated in rural areas accommodating about a quarter of the Mexican population, but 60% of the extreme poor (World Bank, 2004, 2005). In 1997, the Mexican government set up a new nationwide anti-poverty program, named PROGRESA.[2] The program is targeted at the extreme poor in rural areas and is designed as a conditional cash transfer program, meaning that families receive social transfers conditional on the household engaging in a set of behaviors. Program requirements include participation in perinatal care, child health care and immunization, growth and weight monitoring of children, primary and secondary

[1] It is not entirely clear whether the correlation between low parental socioeconomic status (SES) and the lower health status of their children implies a causal relation or that a third factor causes both effects. However evidence increasingly indicates that low parental SES causes poor child health (Currie, 2009).

[2] PROGRESA is an acronym for Programa de Educacion, Salud y Alimentacion (the Education, Health and Nutrition Program). The program was renamed Oportunidades in 2002, but since we use data from the period 1997-1999, we will refer to PROGRESA.

schooling, adult preventive check-ups and nutrition monitoring and supplementation, and finally participation in informational meetings where health and nutrition topics are discussed (pláticas). In this way, the program tries to break the feedback mechanisms that lead to an intergenerational transmission of poverty. By focussing on perinatal care, children's health, nutrition, and schooling, the objective is to enhance poor children's human capital accumulation, and hence future opportunities. By providing monetary resources to families in need and adult preventive care, current poverty is alleviated.[3]

In chapter 2, we analyze the impact of PROGRESA on the participation in and usage of different types of preventive care. Our primary focus is the role of social interactions on the individual or household decision to participate in preventive care. A social interaction effect occurs when an individual participation decision relates to the participation decision of others in an individual's social group. Understanding how social interactions influence behavior is important for policymaking since they could reinforce or offset the direct (financial) incentives given by a social program. Social interaction effects might therefore lead to higher or lower participation rates than otherwise expected and a social program might reach non-targeted individuals and households. In combination with (temporary) direct incentives for behavioral change, social interactions can move a society from a low adoption equilibrium into a high adoption equilibrium (Kremer & Miguel, 2007). Once direct incentives are reduced, important social interaction effects can support the high participation equilibrium. This is especially important for a country like Mexico – characterized by low participation rates in different types of preventive care – that aims at durably increasing participation rates.

We look at the use of deworming drugs, participation of females in cervical screening by means of a Papanicolaou test (abbreviated as pap test), take-up of blood sugar and blood pressure tests by adults, the weight and growth monitoring of children, and child immunization. Despite a high burden of these diseases in Mexico compared to other countries, participation in prevention was low or modest around the start of PROGRESA. Vaccination rates among children were an exception with over 90% vaccination coverage. An analysis of the program effects on health indicates that PROGRESA had a significant positive effect on both adult's and children's health (Barham, 2005, 2011; Gertler, 2000, 2004; Lagarde *et al.*, 2007; Ranganathan & Lagarde, 2012).

[3]The monetary transfers are generally given to the mother of the family, under the implicit assumption that resources managed by women are more likely to be used for schooling, nutrition and other family necessities than money controlled by men.

Estimating peer effects has proven to be challenging because of problems of simultaneity, correlated unobservables and endogenous group membership (Manski, 1993). Early research estimated peer effects as the link between the propensity of the peer group – mostly defined by the researcher based on ethnicity or geographic proximity – to engage in a certain behavior and individual behavior, while controlling for as many group characteristics as possible. Deri (2005) is an example of this approach for health service utilization in Canada, Aizer & Currie (2004) analyze social network effects for participation in publicly funded prenatal care and delivery services. It has been criticized for not overcoming the above-mentioned identification challenges. More recently, researchers use explicit randomization, where a random subset of individuals is 'treated' differently, and this random variation is used as information to identify social interactions more accurately. This line of research exists both for exogenously assigned peer groups and for existing peer groups. An example in health economics of the former is Carrell *et al.* (2011) who analyze fitness outcomes among students at the US Air Force academy who are randomly assigned to squadrons. The problem with this type of study is that peer groups are sometimes created artificially and it is difficult to establish whether estimates are specific to the created situation or are informative for social interactions in the real world. Estimates of peer effects in naturally occurring peer groups are therefore potentially more convincing. Kremer & Miguel (2007), for example, analyze peer effects in the usage of deworming drugs in Kenya using information on household social links. Rao et al. (2012) estimate peer effects in vaccination decisions among US students by using random variation in the ease with which students have access to vaccination locations. Oster & Thornton (2012) look at the role of social interactions in the usage of menstrual cups in Nepal in a school environment.[4]

We follow the promising approach of analyzing social interactions in real world peer groups. We exploit random variation in the eligibility status of individuals and treatment status of localities in PROGRESA as identifying elements in a partial-population setting. As will be discussed below, treatment and control villages are randomly chosen and eligibility status is exogenously determined by the government. This random variation is unrelated to other elements that determine participation and allow us to deal with the identification challenges. Similar methodologies have been applied by Lalive & Cattaneo (2009) and Bobonis & Finan (2009), who analyze the role of peer effects in school enrollment using PROGRESA data, and Dahl *et al.* (2014) who estimate peer effects in

[4]For an overview of research on social interactions in different economic research fields, see e.g. Dahl et al. (2014).

parental leave take-up. In addition, an individual difference in difference approach is used in which we analyze changes in behavior related to the introduction of PROGRESA. The difference in difference approach makes it possible to control for general trends and time invariant heterogeneity. Avitabile (2011) and Barzallo (2011) have done analyzes that look at indirect treatment effects of PROGRESA on health care utilization and health.[5] They find positive spillover effects for participation in cervical screening (Avitabile, 2011) for medical check-ups, and for child and adult health (Barzallo, 2011), while no indirect effect is found for blood pressure and blood sugar tests (Avitabile, 2011). Our approach improves upon their analyzes, since we look at more types of preventive care and are able to attribute the spillover effect to endogenous social interactions. In addition to the identification of endogenous social interactions, we also assess the relative importance of social interaction effects compared to direct financial incentives in changing preventive care participation.

In an influential article, Kremer & Miguel (2007) discuss different channels through which peer effects might work: (1) a desire to imitate the decisions of one's social contacts, (2) signaling[6] or (implicit or explicit) information sharing on benefits, costs or beliefs about preventive care, (3) epidemiological externalities.[7] The latter implies that for infectious diseases, contact with an infected person or location might increase the disease risk. When more social contacts are treated preventively, the risk to contract the disease decreases. While epidemiological effects lead to a negative relation between an individual and his or her peers' use of preventive care, the other channels can give rise to either a positive or a negative effect. Thus, depending on the dominating channel, social interaction effects can be positive or negative. Evidence of the role of social interactions on participation in or usage of preventive care is mixed. Most papers find positive peer effects (e.g. Aizer & Currie, 2004; Deri, 2005; Godlonton & Thornton, 2012; Oster & Thornton, 2012; Munshi & Myaux, 2006; Rao *et al.*, 2007), others find no effect (e.g. Meredith *et al.*, 2013), and even negative effects are found (e.g. Kremer & Miguel, 2007).

Our results indicate that PROGRESA was successful in increasing preventive care usage both among eligible and non-eligible households in treatment villages relative to

[5] With indirect treatment effects, we mean behavioral changes of the non-eligible population in treatment villages.

[6] (Non-)Participation in prevention by a peer might send a signal that prevention yields a higher (lower) level of utility. This might encourage (discourage) participation.

[7] Individuals might in addition also learn how to use a drugs or product from their peers. Given that the preventive care that is analysed in this paper is either easy to apply (taking deworming drugs) or administered by a health professional (vaccination, cancer screening, blood tests), this is less relevant in our setting.

households in control villages. We are able to isolate endogenous social interactions and show that significant positive interaction effects are present for deworming drugs usage, cervical screening, blood pressure tests, and annual child growth and weight monitoring. The magnitude of the peer effects, however, differs across types of prevention. Social interaction effects are especially high for participation in annual growth and weight monitoring of children. Using the information on social interactions, the total treatment effect can be decomposed in a direct effect, related to the financial incentive given to eligible households for complying with PROGRESA requirements, and an indirect social interaction effect. The indirect effect accounts for 10% up to 60% of the total treatment effect for the eligibles, a non-negligible element in explaining the change in preventive health behavior.

The remainder of chapter 2 is structured as follows. Section 2.2 presents a brief discussion of prevention care usage in Mexico, the PROGRESA program, and the data used in the analysis. We elaborate on the research question and design in section 2.3. The main results are presented in section 2.4, followed by a robustness analysis and a conclusion in sections 2.5 and 2.6.

2.2 PROGRESA program and evaluation data

2.2.1 Prevention in Mexico

We look at the use of deworming drugs, participation of females in cervical screening, take-up of blood sugar and blood pressure tests by adults, the weight and growth monitoring of children, and child immunization. Sánchez-Castillo *et al.* (2004) state that traditionally, Mexico's health concerns have been childhood malnutrition and infectious diseases, although the latter has been overtaken by cardiovascular diseases, cancers, and diabetes as the principal causes of death.

In the late nineties, we can state that – except for immunization – Mexico was underperforming in different main aspects of health care. Table 2.1 provides a comparison of some key indicators with respect to the chosen health variables based on OECD and WHO data. Mexico is compared to two OECD countries, its neighboring country, the US, and Chile, which has a similar GDP per capita. Rather than providing a detailed overview of the Mexican health care system in comparison to other countries, the purpose of the provided information is to show that Mexico in 1997 underperformed with respect to the health variables chosen in our analysis and that action was needed.

Table 2.1: OECD data (year 1997) on health indicators from Mexico, Chile and the US

	Mexico	**Chile**	**US**
Doctor consultations per capita	2.3	8.2	3.7
Cervical screening rate (% of females aged 20-69 screened) (data from 2000)	9.7%	64.5%	–
Cervical cancer mortality (deaths per 100,000 females, age standardized)	20.4	15	3.5
Diabetes mortality (deaths per 100,000 individuals, age standardized)	103	30.7	27.7
Circulatory disease mortality (deaths per 100,000 individuals,age standardized)	341.6	322.4	424.7
Infectious disease mortality (deaths per 100,000 individuals, age standardized)	34.3	30	21.5
Neonatal mortality (deaths per 1000 live births)	14	5.7	4.8
Infant mortality (deaths per 1000 live births)	23.8	10	7.2
Low birthweight infants (% of live births)	9.2%	4.8%	7.5%
Immunization rate: measles (% of children immunised)	91.0%	96.0%	91.0%

Note: Unless otherwise stated, the presented data is OECD Health data from 1997.

Despite the existence of a screening program since 1974[8], mortality rates for cervical cancer – which is fully treatable when discovered early – were among the highest in the Americas (Agurto *et al.*, 2004; Lewis, 2004). In the year 2000, the participation rate in Mexico was 10% among females aged 20 to 69 compared to 65% in Chile.[9] The death burden of diabetes – 103 deaths per 100,000 individuals in 1997 – was very high and over three times as large in Mexico than in Chile or the US. The prevalence of hypertension was 33.3% in men and 25.6% in women (Sánchez-Castillo *et al.*, 2004). In 1997, diseases of the circulatory system were as common in Mexico as in Chile, and 25% less common than in the US. Infectious and parasitic diseases accounted for 34 deaths per 100,000 individuals (age standardized rates) in 1997 and the death rate was 10% higher than in Chile and 50% higher than in the US.

Neonatal and infant mortality were double as high in Mexico in 1997 as in Chile and three times as high as in the US.[10] The percentage of children born with low birthweight was 9% in Mexico, or twice as high as in Chile. Moreover, anaemia and micronutrient deficiencies were highly prevalent in Mexico. These conditions can be improved by providing iron and zinc supplements, amongst others (Sepúlveda *et al.*, 2006). Since the introduction of the Mexican universal vaccination program in 1991, vaccination rates among

[8] The national cervical screening program in Mexico offers free screening regardless of age and income and tries to raise awareness among women aged 25 and over.

[9] Cited reasons for non-participation were a low quality of screening, a perceived breach of privacy when the pap test is taken by male doctors, a lack of knowledge, a preference for ignorance since cancer is perceived as deadly, and seeking of medical assistance when the cancer has already entered its late stages rather than screening when feeling healthy (Agurto *et al.*, 2004; Watkins *et al.*, 2002).

[10] Child mortality was high despite important improvements that were made in the period 1980-1997 with a halving of the mortality rate among children under 5 years old (Sepúlveda *et al.*, 2006).

children were high, over 90% and comparable to those in the US and Chile. (Barham, 2005; Sepúlveda *et al.*, 2006).

2.2.2 Program background

In 1997, the Mexican government initiated a large-scale social program aimed at complementing the income of marginalized households in the poorest rural communities and fostering human capital accumulation among children. Monetary transfers were handed out as of 1998 and are conditional on compliance of behavior in two distinct components: 'education' and 'nutrition and health'.

The PROGRESA nutrition and health component is designed to improve health and development from the very start of life. In a first step, PROGRESA aims to decrease the number of low birthweight babies. Low birthweight babies are more susceptible to deficiencies and diseases and run a higher risk of neonatal and infant mortality (Currie, 2009; Gertler, 2000). While some low birthweight babies are able to catch up with their contemporaries, most of them tend to suffer a development disadvantage throughout childhood with potential consequences on future opportunities (Gertler, 2000). PROGRESA imposes pregnant women to have at least 5 prenatal care visits and offers nutritional supplements when needed.

In a second step, young children as well as their lactating mothers are required to attend medical check-ups for growth and weight monitoring and immunization. Children below 24 months are required to attend a check-up at least every two months, while children between 24 and 60 months have an appointment scheduled every four months (Gertler, 2004). Children who lag behind in physical development or are found to be malnourished receive protein and micronutrient supplements, either directly or via their lactating mother. Case & Paxson (2006) argue that poor nutrition during childhood likely affects future cognitive performance. The obligation of growth and weight monitoring for infants combined with the distribution of nutritional supplements potentially has a high pay-off in terms of future human capital accumulation. Immunization policies aim to avoid the occurrence of serious and/or contagious diseases, such as the measles, mumps, tetanus, polio, hepatitis A and B, etc. The Mexican government has a vaccination scheme for children that determines which vaccinations are required at what age.[11]

[11] This is detailed in official norms, such as the Norma oficial Mexicana 031-SSA2-1999 on children's health or the Norma oficial Mexicana 036-SSA2-2002 which brings together prevailing norms and rules on prevention, vaccination, toxic substances etc.

In a third step, attention is paid to the health of adolescents and adults. In order to receive transfers, every family member has to attend a yearly medical check-up. Special attention is paid to reproductive health, family planning, the detection and (preventive) treatment of parasites, of arterial hypertension, of diabetes mellitus, and of cervical cancer (Gertler, 2000). The dangers of these disorders, and the benefits of early detection and treatment as well as health and hygiene habits, are discussed in obligatory pláticas, i.e. village informational meetings for participating households.

PROGRESA is a targeted program. Beneficiaries were identified in two steps (see INSP, 2005). First, highly marginalized rural villages with between 50 and 2,500 inhabitants were selected for sequential entry into the PROGRESA program using a deprivation index. The villages needed to have access to schooling and health care. Next, within the selected villages, poor families were identified. A poverty index score was attributed to all households based on an assessment of their permanent income and household composition. Households with an index score below a certain region-specific threshold were considered poor and could qualify for PROGRESA transfers. Eligibility status and the corresponding rights and benefits were clearly communicated through village-wide assembly meetings. Eligibility status (and non-eligibility status) was awarded for three years and only eligible families that lived in villages where PROGRESA was implemented became potential program beneficiaries. In 1998, PROGRESA was available in 34,400 localities (1.6 million households), and coverage reached as many as 48,700 localities (2.3 million households) in 1999 and 67,500 localities (3.1 million households) in 2001.

2.2.3 Evaluation data and sample selection

An important feature of PROGRESA is that it included an evaluation component. The evaluation design allows the analysis of PROGRESA as a partial-population intervention[12] that is phased in at random. For the evaluation, a subset of 506 localities were selected from across seven states clustered around Mexico city. In October 1997, an initial survey collected socioeconomic information to determine eligibility status of households in all 506 localities.[13] On average, 52% of the households were eligible for PROGRESA, but

[12] A partial-population intervention refers to a design with treated and non-treated (control) clusters. Within the treated clusters only a subset of units are offered the treatment (Baird *et al.*, 2012; Moffitt, 2001).

[13] By July 1999, PROGRESA reclassified a large number of non-eligible households as eligible after complaints that the initial procedure discriminated against the elderly poor who no longer live with their children. The revised households (26% of the evaluation sample) are called the densificado group. However, by August 2000, PROGRESA staff found that many of the newly admitted households had not collected any benefit. Apparently, few densificado households had been notified of their revised eligibility

the percentages vary substantially across localities. Finally, a set of 320 localities were randomly selected as treatment group where PROGRESA was implemented as of April 1998. The remaining 186 communities acted as a control group and were phased in at the start of 2000. The randomization of treatment and control groups has the advantage that it should ensure that both groups are balanced in terms of observable and unobservable characteristics. In addition, multiple surveys are conducted both before and after program implementation. Using appropriate techniques, the effects of PROGRESA can therefore be reliably identified. Behrman & Todd (1999), have thoroughly examined whether pre-program behavior and observable background characteristics are similar in control and treatment groups. They conclude that the randomization procedure worked effectively.

In the evaluation sample, extensive surveys have been carried out to document the effects of PROGRESA. There are two pre-implementation surveys (October 1997 and March 1998) and three post-implementation surveys (October 1998, March 1999 and November 1999) on all 24,000 households in the 506 localities. At the start of 2000, the control group was phased in into the program and additional surveys were conducted. In our analysis, we primarily use the two baseline surveys and the first two post-program surveys.

Each survey contains detailed information on household demographics, socioeconomic status, education, income, expenditures, consumption and health. Not every survey contains the same questions. Questions on the use of health care services and usage are asked in March 1998, October 1998 and March 1999, whereas most pre-program background characteristics are observed in October 1997. Next to household or individual specific information, there are also locality surveys with information on local prices, wages and public and health service availability.

Individual level data on prevention take-up is available for children aged 5 or younger. We construct three preventive care participation indicators: whether a child had attended at least one growth and weight check-up in the past year, whether a child had attended the required number of growth and weight check-ups as imposed by PROGRESA, whether a child complies with the prescribed vaccinations of tuberculosis and measles.[14] Household level data are available on whether or not, in the past year, someone in the household has

status for the program (Buddelmeyer and Skoufias, 2004). Given that we limit our analyses to the first year of the program (March 1998 to March 1999), we consider these households as non-eligible.

[14]There is additional information on vaccination for other diseases, but we focus on take-up of the vaccinations of tuberculosis and measles, since these are infrequent and therefore easily observed. There is one shot at birth for tuberculosis and one shot before age 1 for measles with a renewal around age 6. For tetanus and polio, there are at least four shots before the age of 5 and the data are not recorded accurately enough to follow the vaccination history unambiguously (Barham, 2005).

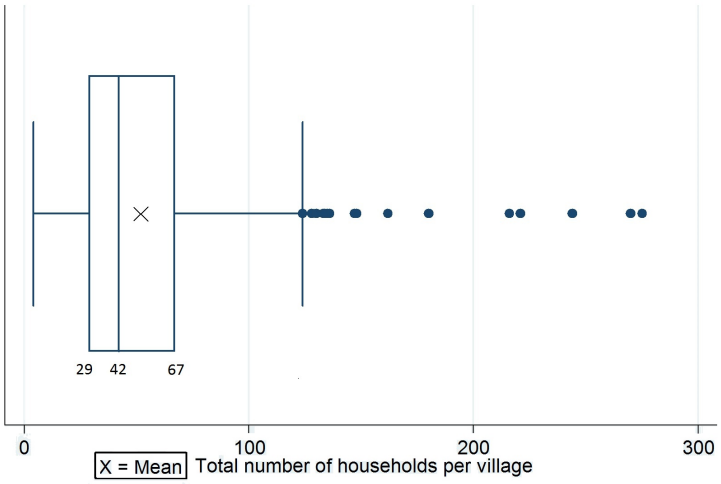
taken deworming drugs, has been screened for high levels of blood sugar or blood pressure. With respect to cervical screening, we construct a variable that indicates compliance with the prevailing Mexican screening norm, i.e. a pap test every three years (after normal test results for two consecutive years). For each indicator of preventive care participation, we have information on pre-implementation and post-implementation participation in prevention, which we combine to construct the change in preventive care take-up. A more detailed description of the construction of the preventive care variables can be found in Appendix 1.

We limit our sample to households for whom we observe their preventive behavior both before and after program implementation. Second, to improve the accuracy of the data, we further restrict the sample to households where the survey is answered by the household head, his or her partner or parents (-in-law) living in the same household. Finally, for the children samples, it should be noted that not all children can be matched perfectly over all surveys. Children who could not be matched unambiguously based on gender, age and household composition are left out of the analysis.

The imposed restrictions entail a risk of sample selection and attrition. Descriptives in Tables 2.11 to 2.17 in Appendix 2 do not show clear signs of sample selection. These tables present descriptive statistics on individual and household characteristics of the entire sample as well as the subsamples by type of prevention used in our empirical analyses. A distinction is made between eligibles and non-eligibles in control and treatment villages. Table 2.11 shows that among the group of non-eligible households, household heads and their partner in control villages are more likely to have started primary education and be able to read and write. Among the group of poor households, the partners of the household heads in control villages are more likely to have started secondary education. Aside from an educational imbalance in favour of control villages, differences between control and treatment villages are minor or non-existent, as one would expect from the random assignment of villages. Among the poor, we find a statistically significant difference in civil status. In treatment villages, couples tend to be married more frequently than in control villages, whereas in control villages couples are more likely to live together outside marriage. Considering couples irrespective of mode of cohabitation, the differences cancel out. The main difference between eligible and non-eligible households can be observed in the marginality index (the criterion for the distinction between both) and other wealth variables.

If we look at the subsamples in Tables 2.12 to 2.15, we observe, in general, similar trends in the subsamples as in the entire evaluation sample. The subsamples are, however, better

Figure 2.1: Distribution of total number of households within a locality (boxplot)



Source: Progresa data

educated, more literate and they have younger and fewer female household heads both for eligibles and non-eligibles in control and treatment villages. Overall, the deviations from the complete sample are limited, which gives us confidence that our estimation results are applicable to the population. The subsamples for growth and weight monitoring and vaccination, i.e. Tables 2.16 and 2.17, contain younger and better educated households than the entire sample. As could be expected, the households in this subsample consist of more couples and have more household members. The differences between control and treatment villages show the same trend as those for the entire sample.

Another important issue to bear in mind is sample attrition. If attrition is correlated with treatment, coefficient estimates could be biased. We define an attrition indicator that takes a value 0 if the household took part in the pre-implementation survey of March 1998 and also answered questions on health related behavior in the post-implementation survey of March 1999. A value 1 is attributed to households who were observed in the March 1998 survey but not in the March 1999 survey. A missing value was attributed to households who did not answer any question on prevention in the March 1998 survey. Attrition is balanced across treatment and control villages with rates of, respectively, 13.6% and 13.3%. Appendix Table 2.18 presents a regression analysis of attrition patterns across all households as well as in the subsamples of eligible and non-eligible households. The results indicate that, when controlling for various household characteristics, attrition

patterns are not affected by treatment status of the villages and are similar for eligible and non-eligible households. The likelihood of attrition is most affected by the number of household members and the age of the household head, i.e. additional household members and an older household head decrease the likelihood of being out of sample. These and other household and village characteristics are controlled for in our empirical analysis.

2.3 Research question and design

The basic idea is that social interactions might play a role in the decision to participate or use preventive care. We use a linear-in-means model to estimate social interactions. In this type of model, peer group average behavior is presumed to influence individual behavior.

We focus on small, naturally occurring peer groups. We assume that the social interactions occur at the locality level, since we lack information on the actual social network of an individual. Thus, the peer group of a household or a child are all other households or all other sampled children, excluding children living in the same household, within the same locality. This choice is justifiable, since rural localities are quite small, with 52 households per village on average, and with fewer than 67 households in 75% of the evaluation localities (see Figure 2.1). Moreover, Adato (2000, p. vi) documents "a common identity in poverty" within the localities. Despite the division created by PROGRESA, there is a perception that everyone is poor, and that "beneficiaries and non-beneficiaries continue to get along with each other fine and 'the same' as before" (Adato, 2000, p. vi). This suggest that social relationships go beyond program eligibility status.

Our research design exploits the quasi-random variation in price for preventive care. As discussed in the previous section, in the PROGRESA program evaluation component, incentives to participate in preventive care are offered to eligible individuals/households in a random subset of villages. Eligibility is determined by a government fixed poverty threshold at the household level. Hence, treatment leads to exogenous variation between treatment and control villages. The main idea is that in treatment villages, incentives given to eligible individuals change their preventive behavior and by extension the average behavior in the entire locality, which we consider to be a relevant peer group. Next changes in peer group behavior might produce changes in individual behavior, including the behavior of non-eligible individuals. Control villages provide a counterfactual situation without government interference. The analysis makes use of the panel data nature of the evaluation surveys to apply a difference in difference design. By focusing on changes in

behavior rather than the actual behavior at a moment in time, we can abstract from time-invariant (un)observed individual, village-level and other heterogeneity.

Our data do not allow us to distinguish between the different channels through which peer effects work. We can however establish whether or not social interactions reinforce PROGRESA's direct incentives.

2.3.1 The model

Let H_{igv} denote the change in preventive care participation and usage between March 1998 and March 1999 of child/family i in peer group g in locality v. The variable can take values 0 (no change), 1 (from non-participation to participation) or -1 (from participation to non-participation). Since peer group and locality are assumed to coincide, we drop the subscript g.[15]

Each locality consist of several eligible (E) and non-eligible (N) individuals. Let us now consider the preventive care decisions of an eligible individual j and a non-eligible individual k within village v:[16]

$$H^E_{jv} = \alpha^E + \beta^E H_{(-j)v} + \delta^E T_v + \varepsilon_{jv} \quad (2.1)$$

$$H^N_{kv} = \alpha^N + \beta^N H_{(-k)v} + \varepsilon_{kv} \quad (2.2)$$

where $H_{(-i)v}$ denotes the peer group average change in preventive behavior excluding individual i. An indicator T_v distinguishes between the randomly assigned groups of treatment villages and control villages. Let $T_v = 1$ denote a treatment village and $T_v = 0$ a control village. Only eligible individuals in village v are offered treatment. The individual specific error term of individual i is given by ε_{iv}. Given randomized treatment assignment, T_v is uncorrelated with observable individual and peer group characteristics as well as the individual error terms, i.e. $E(\varepsilon_{iv}|T_v) = 0$. The direct effect of the PROGRESA program on eligible individuals is captured by δ^E. The parameters of interest are β^E and β^N, which capture the social interaction effects. Eqs. (2.1) and (2.2) capture the idea that an individual's participation decision is influenced by the participation decision made by all other individuals in the relevant peer group. As a robustness check (see section 2.4.6),

[15] The use of village-wide peer groups has the disadvantage that village-specific shocks to the studied beliefs and behavior cannot be controlled for. Some papers infer family connections using information on surnames, see e.g. Angelucci *et al.* (2010). This information is however not publicly available.

[16] For notational simplicity, we do not include observable characteristics.

we analyze an alternative specification in which we consider only the average behavior of eligible peers in the locality. Let us now look at the difference in the expected change in preventive behavior when residing in a treatment versus a control village.

$$E(H^E_{jv}|T_v = 1) - E(H^E_{jv}|T_v = 0) = \delta^E + \beta^E[E(H_{(-j)v}|T_v = 1) - E(H_{(-j)v}|T_v = 0)] \quad (2.3)$$

$$E(H^N_{kv}|T_v = 1) - E(H^N_{kv}|T_v = 0) = \beta^N[E(H_{(-k)v}|T_v = 1) - E(H_{(-k)v}|T_v = 0)] \quad (2.4)$$

As is clear from Eq. (2.3), PROGRESA treatment has a direct program effect (δ^E) and a social interaction effect (β^E) on eligible individuals, whereas it only has a social interaction effect on non-eligible individuals as is shown in Eq. (2.4). Without additional assumptions, it is not possible to distinguish between the direct and indirect effect for eligible individuals. However, β^N can be identified using locality treatment status as an instrument for peer group average preventive behavior. Given that peer groups are composed of eligible and non-eligible households combined, $H_{(-i)v}$ is a weighted average of eqs. (2.1) and (2.2). This leads to the following reduced forms for the peer group average change in preventive behavior:

$$H_{(-j)v} = \mu^E + \tau^E T_v + e_{(-j)v} \quad (2.5)$$

$$H_{(-k)v} = \mu^N + \tau^N T_v + e_{(-k)v} \quad (2.6)$$

where coefficients differ between peer groups of eligibles and non-eligibles. This is a result of a different composition of the peer groups of eligibles and non-eligibles. First, we exclude an individual/household from its own peer group, which leads to different compositions. Second, because of different proportions of eligibles and non-eligibles at the locality level, the fraction of eligible households in the peer group of an eligible household is on average higher than in the peer group of a non-eligible household.[17] Coefficients τ^E and τ^N capture the total program effects on peer group average behavior and $e_{(-i)v}$ represent the group specific error terms.

Inserting eq. (2.6) into eq. (2.2) gives the reduced form of eq. (2.2) and relates the treatment status of the village directly to the change in preventive behavior for non-eligibles.

[17] In localities with a high [low] proportion of eligible households, each eligible household faces a peer group with a high [low] fraction of eligibles. In localities with a high [low] proportion of non-eligible households, each non-eligible household faces a peer group with a low [high] fraction of eligibles. This divergence leads on average to different peer group compositions between eligibles and non-eligibles and to a potentially different effect of PROGRESA on peer group behavior.

$$H_{kv}^{N} = \alpha^{N} + \beta^{N}(\mu^{N} + \tau^{N}T_{v} + e_{(-k)v}) + \varepsilon_{kv} \quad (2.7)$$

$$= \theta^{N} + \pi^{N}T_{v} + e_{kv} \quad (2.8)$$

where π^N can be interpreted as the spillover or "intention-to-treat" (ITT) effect of the PROGRESA program on the preventive behavior of non-eligibles. The social interaction effect β^N is given by the ratio $\frac{\pi^N}{\tau^N}$

For non-eligibles, the indirect effect equals the total treatment effect, as they are not targeted by the program. From a policy point of view, we are not only interested in the presence and magnitude of endogenous social interaction effects, we are equally interested in the direct program effects on the preventive behavior of the targeted population, i.e. δ^E. However, as eqs. (2.1) and (2.3) show, it is not possible to separately identify β^E nor δ^E without additional assumptions. One natural possibility is to assume homogeneous social interaction effects among eligibles and non-eligibles, i.e. $\beta^E = \beta^N$, an assumption implicitly taken by Moffitt (2001) in his seminal work on partial-population designs. Taking this assumption, the total treatment effect can be estimated by regressing the change in preventive behavior on PROGRESA treatment status for the eligible population. In order to see this, let us insert eq. (2.5) into eq. (2.1):

$$H_{jv}^{E} = \alpha^{E} + \beta^{E}(\mu^{E} + \tau^{E}T_{v} + e_{(-j)v}) + \delta^{E}T_{v} + \varepsilon_{jv} \quad (2.9)$$

$$= \theta^{E} + \pi^{E}T_{v} + e_{jv} \text{ with } \pi^{E} = \beta^{E}\tau^{E} + \delta^{E} \quad (2.10)$$

where π^E is the total treatment effect, which can be decomposed in a direct (δ^E) and an indirect effect ($\beta^E\tau^E$). They can be separately identified because of the additional assumption $\beta^E = \beta^N$.

The homogeneity assumption implies that eligibles adapt their preventive behavior to changes in peer group behavior in the same way as non-eligibles do. However, since eligibles get specific information on prevention during obligatory information meetings and receive conditional cash transfers, it is likely that they – in comparison with non-eligibles – rely more on individual signals, than signals from peers. Therefore, we posit that $\beta^E \leqslant \beta^N$ and that the results for $\beta^E = \beta^N$ represent an upper bound on the magnitude of the indirect effects for eligible households.

2.3.2 Identification

Estimating the causal effect of social interactions in a linear-in-means model is challenging given multiple identification issues, such as simultaneity, correlated unobservables, and self-selection into peer groups (Manski, 1993; Moffitt, 2001). The simultaneity of individual behavior relates to the fact that each member in a social group affects every other member. behavioral changes are jointly observed, and it is unclear who affected who. The correlated unobservables problem occurs when not all relevant individual, peer group and environmental characteristics are controlled for. As shown by Moffitt (2001), a partial-population design – whereby the outcome of a randomly chosen subgroup is exogenously altered by some treatment – overcomes this issue. In the framework laid out above, we note that simultaneity is broken down since treatment T_v directly affects preventive behavior of eligible individuals – and by extension peer group average behavior – but it does not directly affect the behavior of non-eligible individuals. Second, because treatment T_v is quasi-random, it is not correlated with observed nor unobserved individual and peer group characteristics. This eliminates bias due to correlated unobservables as is made clear in eqs. (2.7) and (2.9). Finally, the problem of endogenous group membership implies that individuals choose peers with similar tastes, attitudes and preferences, which drives the correlation in decisions and behavior. Bias from this source is addressed by determining peer groups prior to program implementation. Changes in group membership that happen after the implementation of PROGRESA are either a causal result of the treatment or orthogonal to the treatment.

Our identification strategy hinges upon three main assumptions: independence, exclusion and monotonicity.

The independence assumption assures that the reduced forms are consistently estimated. It is implied by randomized treatment. We already established that pre-implementation observable characteristics are fairly balanced with respect to treatment status and sample attrition is not correlated with treatment status. In addition, Table 2.2 shows that pre-implementation differences in preventive behavior between control and treatment villages are in general not statistically significant. One exception is annual child growth and weight monitoring among non-eligible households. Participation in monitoring was 5 percentage points higher in control villages before PROGRESA was implemented.

Second, in order to consistently estimate the size of the social interaction effects using two stage least square, the exclusion assumption implies that the targeting of eligible

households by PROGRESA only affects the preventive behavior of non-eligible children or households through the change in preventive care participation of the peer group. There are some potential concerns. First, non-eligible households might have adapted their preventive behavior to changes in health care supply and quality, which might have developed differently in treatment and control villages. Second, a change in preventive behavior of non-eligible households might be the result of income spillovers. Third, non-eligible households might have misunderstood their eligibility status or anticipated future eligibility and changed their preventive health behavior. Fourth, an improvement in an non-eligible's or his/her peers' education might affect the prevention decision. We discuss the first channel more into detail in section 2.4.4 and the second and third cannel in section 2.4.5. Robustness checks suggests that the exclusion restriction holds. The fourth channel is not likely to play a role in our setting. PROGRESA targeted primary and secondary schooling, while we evaluate the preventive behavior of adults or very young children (below the age of primary schooling). Moreover, since we analyze the first year after program implementation, it is unlikely that the education level of the household head and his or her partner (the main decisionmakers in the household) are affected by the schooling objective of PROGRESA in this time period.

Third, two stage least squares requires the assumption that the PROGRESA reform did not cause any eligible child or household to participate less in prevention (monotonicity). Given the nature of the conditional cash transfer program, this assumption should hold naturally.

Finally, the identification framework assumes implicitly that social interactions occur between eligibles and non-eligibles within a village. The interaction effects are not affected by spillover effects that might occur between for example control and treatment villages that are geographically close. In the literature, this assumption is denoted Stable Unit Treatment Value Assumption (SUTVA) and is very often implicitly or explicitly assumed to estimate social interaction effects. Using GPS data, it is possible to locate the evaluation villages and compute geodesic distances to other villages. We check the validity of SUTVA in section 2.4.5 and conclude that social interaction effects remain positive when relaxing SUTVA, but some estimates lose significance.

2.4 Results

2.4.1 Descriptive evidence

Table 2.2 provides descriptive evidence on the effect of PROGRESA on different types of preventive care. Pre- and post intervention values are reported both for eligibles and non-eligibles averaged over control and treatment villages. Several conclusions can be drawn from Table 2.2.

First, the pre-PROGRESA participation rates for blood sugar test, blood pressure test, and cervical screening are systematically higher among non-eligibles than among eligibles. For the use of deworming drugs, the opposite is true. For prevention among children, pre-implementation participation rates are comparable among eligibles and non-eligibles.

Second, the changes in preventive behavior between pre- and post-PROGRESA levels suggest an increasing participation pattern in preventive care. The trend is especially pronounced for the types of preventive care aimed at adolescents and adults and for growth and weight monitoring at PROGRESA frequency. The fraction of households that is in accordance with the cervical screening norm or took a blood sugar test almost tripled in one year among eligible households in treatment villages, going from 22% to 64%, and it almost doubled in the remainder of the population. Similar effects are observed for child monitoring at PROGRESA frequency. There are also substantial increases in preventive behavior for deworming drugs usage and blood pressure tests. Participation, or usage, increased by 60% to 120% for eligibles in treatment villages and between 40% and 60% in other parts of the population. Annual growth monitoring and child vaccination have high pre-intervention participation rates, over 80% and over 90%, respectively. Hence, the change in behavior is much less pronounced. After program implementation, full participation is almost attained. The increase is fairly equal for vaccination, while for growth monitoring, the change in participation is more pronounced in treatment villages.

Third, in the post-intervention period, we observe that differences between treatment and control villages turn positive and significant for eligibles, except for child vaccination. Also, differences in pre-post levels of preventive behavior turn significant for the eligibles. This is an indication of the total treatment effects of PROGRESA on the beneficiary population and suggests a positive contribution of PROGRESA to health prevention. We can infer from Table 2.2, for example, that the program increased compliance with the cervical screening norm by 19,5 percentage points more among eligibles and by 21 percentage points for blood sugar and blood pressure tests. The program effects implied

Table 2.2: Descriptive evidence on the effect of PROGRESA on participation in prevention

	Eligible				Non-eligible			
	Program	Control	Difference (SD)		Program	Control	Difference (SD)	
Deworming drugs usage								
Drugs usage pre-program	0.511	0.507	0.003	(0.022)	0.439	0.463	-0.024	(0.017)
Drugs usage post-program	0.831	0.719	0.113	(0.018)***	0.636	0.633	0.003	(0.016)
Change in drugs usage	0.321	0.211	0.109	(0.018)***	0.197	0.170	0.027	(0.017)†
Observations	6616	3808			5280	3463		
Cervical screening								
In accordance with screening norm pre-program	0.220	0.247	-0.028	(0.021)	0.270	0.283	-0.014	(0.019)
In accordance with screening norm post-program	0.641	0.474	0.167	(0.026)***	0.542	0.526	0.016	(0.021)
Change in accordance screening norm	0.422	0.227	0.195	(0.019)***	0.272	0.242	0.030	(0.016)*
Observations	6403	3676			5001	3331		
Blood sugar test								
Blood sugar test pre-program	0.232	0.220	0.013	(0.020)	0.314	0.315	0.000	(0.020)
Blood sugar test post-program	0.642	0.420	0.222	(0.024)***	0.539	0.522	0.017	(0.021)
Change in blood sugar test participation	0.409	0.200	0.209	(0.023)***	0.225	0.208	0.017	(0.018)
Observations	6441	3685			5198	3386		
Blood pressure test								
Blood pressure test pre-program	0.355	0.339	0.016	(0.023)	0.459	0.469	-0.010	(0.022)
Blood pressure test post-program	0.769	0.539	0.230	(0.024)***	0.675	0.646	0.029	(0.020)†
Change in blood pressure test participation	0.414	0.200	0.214	(0.022)***	0.216	0.177	0.039	(0.019)**
Observations	6530	3717			5297	3446		
Growth and weight monitoring (yearly)								
Monitoring (at least yearly) pre-program	0.811	0.831	-0.021	(0.024)	0.824	0.873	-0.049	(0.023)**
Monitoring (at least yearly) post-program	0.988	0.946	0.042	(0.010)***	0.962	0.965	-0.003	(0.010)
Change in monitoring (at least yearly)	0.177	0.115	0.062	(0.021)***	0.138	0.093	0.046	(0.020)**
Observations	6518	3773			2148	1554		
Growth and weight monitoring (PROGRESA frequency)								
Monitoring (PROGRESA frequency) pre-program	0.244	0.254	-0.009	(0.010)	0.261	0.263	-0.002	(0.017)
Monitoring (PROGRESA frequency) post-program	0.788	0.674	0.113	(0.010)***	0.659	0.629	0.030	(0.018)*
Change in monitoring (PROGRESA frequency)	0.544	0.421	0.123	(0.014)***	0.398	0.366	0.032	(0.024)
Observations	5194	3056			1651	1208		
Compliance with vaccination scheme								
Vaccination compliance pre-program	0.925	0.929	-0.004	(0.008)	0.927	0.931	-0.004	(0.010)
Vaccination compliance post-program	0.989	0.991	-0.002	(0.002)	0.985	0.987	-0.002	(0.004)
Change in vaccination compliance	0.064	0.062	0.002	(0.008)	0.058	0.056	0.002	(0.010)
Observations	7088	4187			2451	1661		

Note: Mean pre-program values are reported as measured in March 1998. Mean post-program values are reported as measured in October 1998 and/or March 1999. Differences are estimated using OLS regression with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of differences: † p<0.15, * p<0.10, ** p<0.05, *** p<0.01

Source: PROGRESA evaluation data

by the difference in pre-post preventive behavior between control and treatment villages are made even more explicit in Table 2.3. Panel A.1 reproduces the findings of Table 2.2 for eligibles and panel B.1 for non-eligibles. Panels A.2 and B.2 show that the magnitude of the PROGRESA effects are smaller once individual and household characteristics are controlled for. Significance levels remain the same.

Fourth, the pre-post differences in preventive behavior among the non-eligibles are indicative of the spillover effects. A much smaller difference in changes in preventive behavior is observed between non-eligibles in control and treatment villages. With respect to cervical screening for example, the difference in the increase in compliance was 19,5 percentage points among eligibles in treatment and control villages, whereas it is only 3 percentage points among non-eligibles. The differences remain, however, significant for deworming drugs usage, cervical screening, blood pressure tests and annual child monitoring. This suggests the existence of spillover effects and potentially of endogenous social interaction. The effects are small and non-significant for monitoring at PROGRESA frequency and vaccination. In the next subsection, we present the estimates of the social interaction effects.

2.4.2 Estimation of social interaction effects

Table 2.4 reports the main results of the social interaction effects estimates. Panel A provides the IV estimates of the endogenous social interaction effect, β^N, from eq. (2.2). It results from the estimates of the two reduced-form equations, eqs. (2.6) and (2.8). Panel B reports the effects of the former, while the latter is presented in panel C. Taking deworming drugs usage as an example (column 2), the results should be read as follows: for each non-eligible household who lives in a treatment village, the usage rate in the peer group increases on average by 6.7 percentage points. This increase in peer group usage leads on average to a 2.7 percentage point increase in the usage of non-eligible households. The peer group responsiveness is generally stronger than the behavioral change of non-eligibles, because the peer group partly consists of eligibles, whose behavioral change is financially incentivized. The relation between the peer group responsiveness and the household responsiveness gives the social interaction estimator. As the household responsiveness increases (decreases) relative to the peer group responsiveness, this translates into a higher (lower) social interaction parameter.

Table 2.3: Participation in prevention: treatment and spillover effects among eligibles and non-eligibles

Dependent variable:	Deworming drugs usage	Cervical screening	Blood sugar test	Blood pressure test	Monitoring (yearly)	Monitoring (Progresa)	Vaccination compliance
A. Eligibles							
A.1 OLS regression without controls							
PROGRESA treatment	0.109***	0.195***	0.209***	0.214***	0.062***	0.123***	0.002
(Standard error)	(0.018)	(0.019)	(0.023)	(0.022)	(0.021)	(0.024)	(0.008)
A.2 OLS regression with individual and household controls							
PROGRESA treatment	0.109***	0.189***	0.203***	0.211***	0.065***	0.137***	0.003
(Standard error)	(0.017)	(0.018)	(0.022)	(0.021)	(0.019)	(0.023)	(0.009)
Observations	10235	9963	9942	10057	10127	8158	10806
B. Non-eligibles							
B.1 OLS regression without controls							
PROGRESA treatment	0.027†	0.030*	0.017	0.039**	0.046**	0.032	0.003
(Standard error)	(0.017)	(0.016)	(0.018)	(0.019)	(0.020)	(0.031)	(0.010)
B.2 OLS regression with individual and household controls							
PROGRESA treatment	0.024†	0.027*	0.015	0.037**	0.041**	0.031	0.005
(Standard error)	(0.016)	(0.015)	(0.018)	(0.018)	(0.019)	(0.031)	(0.010)
Observations	8650	8245	8493	8651	3655	2825	3948

Note: Coefficient estimates from OLS regressions are reported with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of coefficients: † p<0.15, * p<0.10, ** p<0.05, *** p<0.01. For all regressions, controls include: number of household members (dummies, capped at a maximum of 6 or more), number of children (dummies, capped at a maximum of 6 or more), fraction of poor families/children in the village, household marginality index, characteristics of floor and roof of the house, agricultural land owner, household head civil status (in dummies: married, partner but not married, separated, widow), household head age, household head characteristics (dummies for: literacy, primary education, secundary education, missing education info, speaking indigenous language), partner of household head age, partner characteristics (dummies for: literacy, primary education, secundary education, speaks indigenous language). For cervical screening, the regression controls for the number of females aged 16 or more in the household (dummies, capped at a maximum of 6 or more). For vaccination and growth monitoring, the regression controls for the age of the child in March 1998, before program implementation.

Table 2.4: IV and OLS estimates of endogenous peer effects among non-eligibles

Dependent variable:	**Deworming drugs usage**		**Cervical Screening**		**Sugar test**		**Pressure test**		**Monitoring (yearly)**		**Monitoring (Progresa)**		**Vaccination compliance**	
A. IV estimates (Dependent variable: Changes in individual values H^N_{kv}; Coefficient of interest: β^N)														
Changes in peer group mean values ($H_{(-k)v}$)	0.398**	0.383*	0.296**	0.293**	0.165	0.149	0.328***	0.334***	0.704***	0.675***	0.380	0.362	0.672	1.447
(Standard error)	(0.184)	(0.212)	(0.118)	(0.124)	(0.154)	(0.151)	(0.118)	(0.120)	(0.189)	(0.197)	(0.302)	(0.281)	(1.754)	(1.907)
Individual and household controls	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Peer group controls	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Observations	8743	8650	8332	8245	8584	8493	8743	8651	3694	3619	2856	2794	3979	3908
B. OLS Peer group regression (Dependent Variable: Changes in peer group mean values $H_{(-k)v}$; Coefficient of interest: τ)														
PROGRESA treatment village (T_v)	0.067***	0.059***	0.101***	0.095***	0.104***	0.101***	0.120***	0.115***	0.065***	0.062***	0.086***	0.094***	0.004	0.004
(Standard error)	(0.014)	(0.013)	(0.015)	(0.014)	(0.017)	(0.016)	(0.017)	(0.016)	(0.020)	(0.017)	(0.023)	(0.022)	(0.007)	(0.007)
Kleibergen-Paap F statistic	22.66	19.80	45.48	44.74	36.68	39.48	49.23	50.86	10.75	13.64	13.86	17.34	0.35	0.36
C. OLS Individual regression (Dependent Variable: Changes in individual values H^{NE}_{kv}; Coefficient of interest: π^N)														
PROGRESA treatmentvillage (T_v)	0.027†	0.023	0.030*	0.028*	0.017	0.015	0.040**	0.038**	0.046**	0.042**	0.033	0.034	0.003	0.006
(Standard error)	(0.017)	(0.016)	(0.016)	(0.015)	(0.018)	(0.017)	(0.019)	(0.018)	(0.020)	(0.018)	(0.031)	(0.031)	(0.010)	(0.010)

Note: Coefficient estimates from OLS and 2SLS IV regressions are reported with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of coefficients: † p<0.15, * p<0.10, ** p<0.05, *** p<0.01. The Stock-Yogo (2005) critical value for the Kleibergen-Paap weak identification F statistic at 10% maximal IV size is 16.38 and 8.96 at the 15% maximal IV size. For all regressions, controls include: number of household members (dummies, capped at a maximum of 6 or more), number of children (dummies, capped at a maximum of 6 or more), fraction of poor families/children in the village, household marginality index, characteristics of floor and roof of the house, agricultural land owner, household head civil status (in dummies: married, partner but not married, separated, widow), household head age, household head characteristics (dummies for: literacy, primary education, secundary education, missing education info, speaking indigenous language), partner of household head age, partner characteristics (dummies for: literacy, primary education, secundary education, speaks indigenous language). In addition, peer group averages are included for household members and children, household poverty index, household head and partner characteristics and a village dummy for extreme poverty (as opposed to very poor). For cervical screening, the regression controls for the number of females aged 16 or more in the household (dummies, capped at a maximum of 6 or more). For vaccination and growth monitoring, the regression controls for. the age of the child in March 1998, before program implementation.

Source: PROGRESA evaluation data

The results in Table 2.4 indicate that social interaction effects are positive and significant for four types of preventive care, i.e. deworming drugs usage, blood pressure test, cervical screening, and annual child monitoring. The magnitude of the social interaction effect varies across the different types of prevention. They are especially important for annual weight and growth monitoring of children.[18] For vaccination compliance, no effects are found. For participation in blood sugar tests and child monitoring at PROGRESA frequency, minor positive effects are found, but estimated imprecisely.

Estimates are reported both with and without controlling for individual and household characteristics and peer group effects. The estimates of the social interaction effect are robust to the inclusion of control variables, which is a good sign for our identification strategy. One exception is the social interaction effect for vaccination compliance. However, social interactions for vaccination compliance are unreliably estimated due to a weak first stage. We test for potential weakness of the instrumental variable using the Kleibergen-Paap Wald F statistic, which is robust to clustered standard errors. The F-statistic shows that treatment status as instrument is – except for vaccination compliance – not weak, which lends credibility to our baseline estimates.

What can we learn from the results in Tables 2.2 to 2.4? Immunization of children below 5 years old against tuberculosis and measles has been generally adopted before PROGRESA was set up and compliance among this group of children increased further as they aged. Vaccination compliance was not different between eligibles and non-eligibles in control and treatment villages. Table 2.3 shows no PROGRESA effect among eligibles or non-eligibles. The lack of direct impact of PROGRESA explains the absence of social interaction effects.

Participation in annual growth and weight monitoring of children was high before PROGRESA started and increased to almost full participation one year later. Pre-intervention monitoring according to PROGRESA's guidelines was much lower. Less than a third of all children below 5 years were monitored regularly, but compliance more than tripled among treated eligibles after one program year. It increased slightly less among the other groups. The increase in child monitoring on an annual basis and according to PROGRESA frequency is 6.2 and 12.3 percentage higher among the eligibles in treatment villages than in control villages, respectively, providing evidence of a PROGRESA treatment effect. The

[18] Annual participation in growth and weight monitoring was already high before PROGRESA was introduced and increased further among the eligibles through the financial incentives. It is possible that non-participation became socially disapproved and the desire to conform higher than for other types of preventive care.

results in Table 2.4 establish the presence of social interactions for annual monitoring, but not for monitoring at PROGRESA frequency.

With respect to adolescent and adult preventive health care, the patterns are similar, but the actual magnitude of the effects differ. Despite a relatively high prevalence of cervical cancer and diabetes in Mexico (see Section 2.2.1), participation rates for cervical screening and blood sugar tests were low. The pre-program participation for households in our sample was below 25% for eligibles and a little above 25% for non-eligibles. Take-up of blood pressure tests and the usage of deworming drugs was higher and fluctuated between 35% and 50%. In Tables 2.2 and 2.3, we observe a large increase in preventive take-up in all layers of the population for all four types of prevention. The increase among eligibles in treatment villages is, however, much more pronounced, allowing us to conclude that there was an important direct effect of the stimuli to attend preventive check-ups. The change in behavior among non-eligibles was also systematically higher in treatment villages than in control villages, the difference is, however, small, and not significant for blood sugar tests. Our social interaction estimates in Table 2.4 suggest that the spillover effects from eligibles to non-eligibles are the result of social interactions, with significant effects for deworming drugs usage, participation in cervical screening and take-up of blood pressure tests. For blood sugar tests, the social interaction effect is not significantly different from zero once control variables are added.

We conclude that social interactions transmit policy incentives to enhance preventive behavior among targeted individuals, to non-targeted individuals. It is not entirely clear which channels are driving the social interaction effects, but the results are consistent with positive imitation effects and (implicit or explicit) information sharing on benefits. For deworming drugs usage, these positive effects outweigh potential negative externality effects.

2.4.3 Direct versus indirect effect

Table 2.5 presents the decomposition of the total treatment effect of PROGRESA on eligibles and non-eligibles in a direct and an indirect effect. Remember that the homogeneity assumption implies that, most likely, the estimates of the direct and indirect effect are situated at their lower and upper bound, respectively. The analysis is performed for all types of preventive care except vaccination, since we have found no indication of a direct or indirect effect for child immunization. Panel A shows the results for the eligibles and panel B for the non-eligibles. Only the indirect effect plays for the latter.

Table 2.5: Decomposition of the total treatment effect of PROGRESA

Dependent variable:	Deworming drugs usage	Cervical screening	Blood sugar test	Blood pressure test	Monitoring (yearly)	Monitoring (Progresa)
A. Eligibles						
1. Total treatment effect	0.109***	0.187***	0.202***	0.219***	0.066***	0.138***
(Standard error)	(0.016)	(0.017)	(0.021)	(0.020)	(0.019)	(0.023)
2. Social interaction parameter	0.383*	0.293**	0.149	0.334***	0.675***	0.362
(Standard error)	(0.212)	(0.124)	(0.151)	(0.120)	(0.197)	(0.281)
3. PROGRESA effect on peer group	0.072***	0.130***	0.129***	0.150***	0.061***	0.116***
(Standard error)	(0.015)	(0.015)	(0.018)	(0.019)	(0.018)	(0.021)
4. Indirect effect (2 x 3)	0.028*	0.038**	0.019	0.050***	0.041**	0.042
(Standard error)	(0.016)	(0.017)	(0.020)	(0.019)	(0.017)	(0.033)
5. Direct treatment effect (1 - 4)	0.081***	0.149***	0.183***	0.169***	0.025***	0.095***
(Standard error)	(0.012)	(0.013)	(0.019)	(0.015)	(0.008)	(0.016)
Indirect effect as % of total effect	25.69%	20.32%	9.41%	22.83%	62.12%	30.43%
B. Non-eligibles						
1. Total treatment effect	0.023	0.028*	0.015	0.038**	0.042**	0.034
(Standard error)	(0.016)	(0.015)	(0.017)	(0.018)	(0.018)	(0.031)
2. Social interaction parameter	0.383*	0.293**	0.149	0.334***	0.675***	0.362
(Standard error)	(0.212)	(0.124)	(0.151)	(0.120)	(0.197)	(0.281)
3. PROGRESA effect on peer group	0.059***	0.095***	0.101***	0.115***	0.062***	0.094***
(Standard error)	(0.013)	(0.014)	(0.016)	(0.016)	(0.017)	(0.022)
4. Indirect effect (2 x 3)	0.023	0.028*	0.015	0.038**	0.042**	0.034
(Standard error)	(0.016)	(0.015)	(0.017)	(0.018)	(0.018)	(0.031)
5. Direct treatment effect (1 - 4)	–	–	–	–	–	–
(Standard error)	–	–	–	–	–	–

Note: Coefficients in rows 1 are the result of estimating eqs. (2.8) and (2.10). Coefficients in rows 2 come from Table 2.4. Coefficients in rows 3 are the result of estimating eqs. (2.5) and (2.6). All regressions have control variables as specified in Table 2.4. Rows 1 to 3 have robust standard errors that allow for correlation of disturbance terms within localities. Standard errors in row 4 are computed using the delta method. Standard errors in row 5 are robust and allow for correlation of disturbance terms within localities. They are obtained using an OLS regressions constraining $\beta^E = \beta^N$. Significance levels of coefficients: † p<0.15, * p<0.10, ** p<0.05, *** p<0.01.
Source: PROGRESA evaluation data

Row 1 in panel A shows the estimation of the total treatment effect as laid down in eqs. (2.8) and (2.10), while row 4 shows the indirect effect and row 5 the direct effect. The latter is only relevant for eligibles and is always significant. It varies from a 2.5 percentage points increase in annual growth monitoring to a 18.3 percentage points increase in the take-up of blood sugar tests. The indirect effect is smaller and increases participation rates in prevention by 1.5 to 5 percentage points. It is, in general, smaller among the non-eligibles than among the eligibles.

If we calculate the share of the indirect effect in the total treatment effect, we find that social interactions contribute to 20% of the total change in cervical screening. It increases up to around 25% for deworming drugs usage and 22% for blood pressure tests and 30% and 62% for child growth and weight monitoring, respectively at PROGRESA frequency and on an annual base. At least for these types of preventive care, it appears that social interactions explain a non-negligible part of the change in preventive behavior

that is observed after the introduction of PROGRESA. It is striking that for blood sugar tests, the total treatment effect on the target population is large, but that this is almost entirely due to the direct incentives. It has not spilled over to non-eligibles.

2.4.4 Health care supply and quality

Changes in preventive behavior can be a response to changes in health care supply and quality, which we chose to neglect until now. If supply and quality developed differently in treatment and control villages, e.g. because of the implementation of PROGRESA, this can have an effect on the social interaction estimates. Agurto *et al.* (2004), for example, conclude from focus groups and interviews that, amongst others, time costs, unfriendliness of providers, inadequacy of counseling, and poor quality material and instruments are important barriers to participation in cervical screening in Mexico and other Latin American countries. Similar effects could play for other types of preventive care. Changes in offered services, quality, or prices could thus perfectly lead to the observed changes in health demand and behavior. The differential increase in preventive health behavior between treatment and control villages might be the result of improvements in health supply or quality in treatment villages relative to control villages or to a change in (time) costs to attend medical services.

PROGRESA survey data contain information that makes it possible to test this mechanism. Pre- and post-intervention information is available on the type of health care providers that are located in or around the locality, the type of services offered, the opening time, the perceived quality (sufficient staff and material, clear explanation of problem, quality of doctors and quality of nurses), and waiting time.[19] Information on health care providers and services are at the locality level, the other data are recorded at the household level. However, we average the household level information at the locality level, since only a limited number of households have provided the information and restricting our subsamples further to this group would eliminate many observations and potentially create bias.

[19] We construct the following variables: a dummy variable that indicates whether any provider (hospital, doctor, health aid or midwife) was available in the locality. A variable, ranging from 0 to 7, indicating the number of services available in the locality (pre-natal care, delivery care, baby care, immunization, family planning service, hospitalization, diarrhea care). Opening time in hours per week. An index score, ranging from 0 to 1 for the availability of staff and equipment, based on 4 yes/no questions (has medical centre sufficient doctors?, nurses?, medication?, material?). An index score, ranging from 0 to 1 for the quality of doctors, based on 4 yes/no questions (is doctor respectful?, prepared?, responsible? and confident?). A similar quality index variable for nurses. A dummy indicating whether doctors provide clear information. Waiting time is indicated in minutes per visit.

Table 2.6: Descriptive evidence on health supply, quality and waiting time

	Pre-program				Post-program			
	Treatment	Control	Difference (SD)		Treatment	Control	Difference (SD)	
Health care provider present (0=No, 1=Yes)	0.959	0.945	0.014	(0.022)	0.960	0.968	-0.007	(0.017)
Services available (0 to 7)	2.727	2.958	-0.232	(0.217)	3.556	3.368	0.188	(0.235)
Opening time (hours per week)	10.443	10.233	0.210	(0.267)	9.233	9.192	0.041	(0.226)
Availability staff and equipment (0 to 1)	0.577	0.561	0.015	(0.017)	0.575	0.586	-0.012	(0.016)
Quality of doctors (0 to 1)	0.972	0.976	-0.004	(0.003)	0.960	0.957	0.002	(0.008)
Quality of nurses (0 to 1)	0.959	0.960	-0.001	(0.005)	0.952	0.956	-0.003	(0.007)
Clear explanation given (0=No, 1=Yes)	0.986	0.978	0.007	(0.003)**	0.979	0.985	-0.006	(0.003)*
Waiting time (minutes per visit)	55.418	59.054	-3.636	(2.416)†	55.769	58.958	-3.189	(1.871)*

Note: Mean pre-implementation values are reported as measured in March 1998. Mean post-implementation values are reported as measured in October 1998 and/or March 1999. Differences are estimated using OLS regression with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of differences: † p<0.15, * p<0.10, ** p<0.05, *** p<0.01

Source: PROGRESA evaluation data

Table 2.7: Social interaction estimates when controlling for changes in health supply characteristics β^N

	1. Baseline	**2. Health supply**	**3. Health supply quality and time**
Deworming drugs usage	0.383*	0.408**	0.468**
(Standard error)	(0.212)	(0.200)	(0.188)
Cervical screening	0.293**	0.308**	0.313**
(Standard error)	(0.124)	(0.122)	(0.126)
Blood sugar test	0.149	0.162	0.235*
(Standard error)	(0.151)	(0.148)	(0.127)
Blood pressure test	0.334***	0.359***	0.389***
(Standard error)	(0.120)	(0.114)	(0.110)
Monitoring (yearly)	0.675***	0.677***	0.656***
(Standard error)	(0.197)	(0.194)	(0.207)
Monitoring (PROGRESA)	0.362	0.340	0.346
(Standard error)	(0.281)	(0.286)	(0.282)

Note: Social interaction estimates from IV regressions are reported with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of coefficients: † p<0.15, * p<0.10, ** p<0.05, *** p<0.01. All regressions have control variables as specified in Table 2.4.
Source: PROGRESA evaluation data

Table 2.8: First priority in spending additional monthly household resources (% of households)

	All households	Non-eligible households
Food consumption	77.0%	74.4%
Debt payment and saving	6.6%	7.8%
Housing expenses	5.4%	6.1%
Clothing and shoes	4.9%	4.7%
Investments in agriculture (seeds, animals, tools)	3.2%	3.9%
Medication	1.6%	1.9%
School supplies	1.0%	0.9%
Other expenditures (alcohol, toys, entertainment)	0.2%	0.3%

Source: PROGRESA evaluation data

Table 2.9: Robustness results of social interaction estimates β^N

	1. Baseline	2. Gifts received	3. Anticipation effects	4. Environment and public health	5. Distance controls	6. Distance sample restrictions	7. Two instruments	8. Alternative peer group
Deworming drugs usage	0.383*	0.340†	0.359†	0.402*	0.368†	0.244	0.454**	0.192†
(Standard error)	(0.212)	(0.218)	(0.241)	(0.216)	(0.226)	(0.394)	(0.191)	(0.133)
Cervical screening	0.293**	0.257**	0.281**	0.297**	0.321***	0.198	0.286**	0.154**
(Standard error)	(0.124)	(0.130)	(0.130)	(0.124)	(0.113)	(0.178)	(0.121)	(0.178)
Blood sugar test	0.149	0.125	0.111	0.178	0.127	0.095	0.199†	0.075
(Standard error)	(0.151)	(0.155)	(0.163)	(0.145)	(0.156)	(0.182)	(0.138)	(0.080)
Blood pressure test	0.334***	0.326***	0.247*	0.363***	0.282**	0.386***	0.351***	0.181**
(Standard error)	(0.120)	(0.120)	(0.147)	(0.113)	(0.128)	(0.123)	(0.117)	(0.080)
Monitoring (yearly)	0.675***	0.691***	0.691***	0.603***	0.640***	0.779***	0.701***	0.651***
(Standard error)	(0.197)	(0.192)	(0.255)	(0.195)	(0.206)	(0.272)	(0.171)	(0.257)
Monitoring (PROGRESA)	0.362	0.351	0.265	0.370	0.394	0.486	0.460*	0.276
(Standard error)	(0.281)	(0.282)	(0.385)	(0.279)	(0.284)	(0.378)	(0.253)	(0.243)

Note: Social interaction estimates from IV regressions are reported with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of coefficients: † p<0.15, * p<0.10, ** p<0.05, *** p<0.01. All regressions have control variables as specified in Table 2.4.

Source: PROGRESA evaluation data

In Table 2.6, the pre- and post-intervention values are indicated for treatment and control villages as well as the differences. Table 2.6 shows that almost all localities have at least one health care provider (which was a program requirement). The number of services is similar in treatment and control villages and has increased slightly after PROGRESA started. There are two main conclusions. First, the descriptive evidence shows that supply did not change in any important way after program implementation. Second, differences between control and treatment villages are minor. This is a first indication that the changes in preventive behavior probably do not results from changes in health care supply or perceived quality.

Second, we report social interactions estimates in Table 2.7 when controls for health supply, health care quality and waiting time are subsequently added. Column 1 reproduces the baseline results, for all prevention types except vaccination where social interaction effects are non-existent. Controlling for health provision characteristics, the social interaction estimates are in line with or somewhat higher than the baseline results.[20] Overall, we conclude that differences between control and treatment villages are limited and the inclusion of control variables for health supply and quality do not affect our results.

2.4.5 Alternative explanations

In order to support the validity of the results shown in Table 2.4, we address in Table 2.9 three alternative channels, brought up in section 2.3.2, that might have generated the observed changes in preventive health behavior of non-eligible households in treatment villages. The first column reproduces the baseline estimates.

First, changes in preventive behavior of non-eligible households might be the result of income spillovers. PROGRESA provides monetary transfers to program eligible households who comply with program requirements. There is a possibility of monetary transfers from eligible to non-eligible households through gifts, loans or increased spending of eligible households in shops of non-eligible households in their locality. Part of the income spillovers can be used to increase medical consumption. Adato (2000) concludes from focus group research that sharing of benefits by eligible households is rare, since benefits are perceived as small and used primarily to finance schooling costs (Lalive & Cattaneo, 2009), increase food consumption and food quality, and buy clothing (Bobonis, 2004; Hoddinott & Skoufias, 2004). The increase in expenditures by eligible households might indirectly

[20] Especially the effects of doctors who provide a clear explanation and doctor quality play an important role. However, none of the new point estimates is significantly different from the baseline estimates.

benefit non-eligible households if the additional expenditures are realized in shops owned by non-eligibles. This is, however, not the case since only 20 out of the 506 villages have a local supermarket or street market (Lalive & Cattaneo, 2009). Nonetheless, Angelucci & De Giorgi (2009) present evidence that non-eligible households in treatment villages have received more gifts and loans since PROGRESA was rolled out. The additional resources are used to increase food consumption levels but are not sufficient to cover the increase in food expenditures. It appears that little additional money is available for increased medical consumption. Moreover, it seems that medical expenses are not prioritized. In the March 1998 pre-implementation survey, it is asked what the top priority would be to spend additional monthly household resources. Medication was among the possible answers, and was prioritized by only 2% of the households (see Table 2.8). Food is preeminently given the highest priority, in accordance with the results of Angelucci & De Giorgi (2009), Bobonis (2004) and Hoddinott & Skoufias (2004).

Given the limited evidence of income spillovers in treatment villages and the fact that increases in financial resources are primarily used to finance food consumption, we argue that this channel provides no good alternative explanation for potential social interaction effects in prevention. Nonetheless, we provide a test for income spillovers. We add information (from the post-implementation survey in October 1998) to the baseline specification on the amount of monetary gifts that a household has received and we add dummies for receiving food and clothes through in kind gifts as proxies for income spillovers. Column 2 in Table 2.9 shows that the inclusion of gift variables does not affect the baseline estimates.

PROGRESA transfers not only lead to a potential increase of monetary resources of non-eligible households, they also lead to an increase in income at the peer group level, again potentially affecting the preventive behavior. A regression analysis of pre-implementation preventive behavior on the household poverty index (which captures permanent income corrected for household composition), the peer group average poverty index and additional household and peer group control variables (the same control variables as used throughout our main analysis, see Table 2.4), reveals that peer group average income does not significantly affect preventive behavior (results available on request). There is one notable exception. In March 1998, deworming is positively correlated with household income and negatively correlated with peer group average income in our sample. Both coefficients are highly significant ($p<0.01$). One explanation is that richer communities might have better public health provisions and a lower risk of parasite infections. The negative correlation might lead to an underestimation of social interaction effects for

deworming. For the pap test, we find a positive income effect at the peer group level, but only borderline significant (p=0.07). This was not expected, as the pap test is free of charge for all women, so that income should not play an important role, unless social acceptance of the test is different according to the average income level of the community. We controlled for pre-implementation peer group average income throughout our empirical analysis.

Second, non-eligible households might have misunderstood their eligibility status or anticipated future eligibility and changed their preventive health behavior. This is unlikely to be the case, since households were notified clearly about their eligibility status. Moreover, eligibility was awarded until at least November 1999 and during this period non-eligible or new households were not able to attain eligibility status, irrespective of income or behavior (Angelucci & De Giorgi, 2009). We test for anticipation effects by removing the 25% non-eligible households that are closest to the poverty cut-off point, and hence most likely to be influenced by anticipation effects. If anticipation effects drive our results, the removal of the poorest non-eligibles would reduce the social interaction effects drastically. The results in column 3 show that this is not the case. None of the new point estimates are significantly different from the baseline coefficients. The most notable decreases in coefficient value are observed for blood pressure tests and growth monitoring at PROGRESA frequency.

Third, it might be the case that environmental or public health factors have driven the change in prevention participation among the non-eligibles in treatment villages. The difference in difference approach captures time invariant heterogeneity. In column 4, we add dummies for natural disasters (drought, flood, earthquake, frost, pest and a residual category) that occurred between April 1998 and March 1999. The information is household specific, but we also add peer group averages. Moreover, we add dummies to control for localities with sewer systems and public water networks. In localities with better public health provisions, prevention participation may evolve differently, especially with respect to infections by parasitic diseases which are linked to public hygiene and sanitation (Meredith *et al.*, 2013). The estimates presented in column 4 show no important deviations from the baseline results.

Fourth, we assume that social interactions occur between eligibles and non-eligibles within a village and not across villages (SUTVA). Spillover effects across villages can originate from shared health care suppliers, cooperations among neighboring localities, interactions among villagers etc. We test SUTVA in two ways. The analysis presented in

column 5 in Table 2.9 adds distance controls to the baseline regression. These controls include the shortest geodesic distance to a treatment village, the number of treatment villages within a 5 km and within a 10 km radius and the total number of villages within a 5 km and within a 10 km radius.[21] Adding distance controls hardly changes the coefficient estimates. In column 6, we perform the baseline analysis, but on the subsample of localities without another treatment village within a 5 km radius. This reduces our sample by almost a third, which leads to larger standard errors. The social interaction estimates for deworming, cervical screening and blood sugar test decrease by a third, whereas for the other types of prevention the estimates increase by 15% to 30%. As a consequence of the decrease in coefficient values and the larger standard errors, estimates for deworming and cervical screening are no longer significant. To conclude, our results are mixed with respect to relaxing SUTVA, social interaction effects remain positive, but some estimates lose significance.

2.4.6 Alternative specifications

As a last part of our robustness analysis, we analyze two alternative specifications. First, we add the fraction of eligible individuals in treatment villages as a second instrument, next to village treatment status. We have not used the share of eligibles in treatment villages as instrument throughout the main analysis, since it may not be exogenous if there is any sorting of families in and out of the village based on unobservable characteristics of the households or villages. This concern can be alleviated somewhat by fixing the share of eligibles at the pre-intervention level, which is what we do. The main reason for adding a second instrument is to increase precision. The results in column 7 in Table 2.9 show that standard errors decrease only moderately and that the coefficient estimates are in line with the baseline.

Second, we estimate the social interaction effect β^N but with a different peer group. We do not treat the entire village as the relevant peer group for a non-eligible household, but only the subgroup of eligibles. The results are shown in column 8 of Table 2.9. The social interaction estimates decrease drastically, except for annual child monitoring. This result follows logically from the choice of peer group and could have been anticipated from Table 2.2. If the peer group consists only of eligible households, the peer group average change in preventive behavior is much more pronounced and stronger correlated

[21] On average, 30% and 69% of the localties have at least one treatment village within a 5 km and 10 km radius, respectively; 42% and 80% of the localties have at least on other village within a 5 km and 10 km radius, respectively. The shortest geodesic distance to a neighboring treatment village is on average 9.4 km .

to the village treatment status. The non-eligible household responsiveness on the other hand does not change. Thus, the relative decrease of the household responsiveness to the peer group responsiveness translates into a lower social interaction parameter. This effect plays less for annual child monitoring since the change in behavior among eligibles is not much above the change in behavior among non-eligibles, which means that the peer group responsiveness does not alter much when opting for the alternative definition. While the coefficient values change quite drastically, the sign and significance do not. The choice of peer group is important, especially with respect to the magnitude of the social interaction effects (and thus the decomposition between direct and indirect effects), but not with respect to its existence and reinforcing role.

2.5 Conclusion

Individual participation in preventive health care may depend on preventive health behavior in the peer group of the individual. This chapter analyzes the importance of social interactions in the context of new social policies in Mexico that aim to increase health care usage among a targeted subgroup of the population. We followed the promising approach of analyzing social interactions in real world peer groups. We exploited the partial-population design with random variation in eligibility status of households and in treatment status of localities in PROGRESA for the identification of social interactions.

Results indicate that PROGRESA was successful in increasing preventive care usage among the eligible households. Non-eligible households in treatment villages have also changed their preventive health behavior more than their counterparts in control villages, providing evidence of spillover effects. We were able to identify endogenous social interactions – under relatively weak assumptions – and showed that social interaction effects are present for deworming drugs usage, cervical screening, blood pressure tests and annual child growth and weight monitoring. No social interactions are found for immunization of children and for blood sugar tests. The magnitude of the social interaction effects differs across types of prevention. The results are robust to the inclusion of health supply, quality and waiting time controls. The social interaction effects remain when we consider income spillovers, anticipation effects and effects from environmental shocks or differences in public sanitation. Relaxing the SUTVA condition leads to mixed conclusions, social interaction effects remain positive, but some estimates lose significance.

Using the information on social interactions, the total treatment effect can be decomposed in a direct effect, related to the financial incentive given to eligible households for

complying with PROGRESA requirements, and an indirect effect. The total treatment effect indicates that participation in prevention among eligibles increased as much as 20 percentage points for cervical screening, blood sugar and blood pressure tests, around 14 percentage points for growth monitoring at PROGRESA frequency, 11 percentage points for deworming drugs usage and 7 percentage points for annual monitoring. The latter started with a pre-program participation rate well above 80%. The indirect effect due to social interactions accounts for 10% up to 60% of the total treatment effect for the eligibles, i.e. a non-negligible share.

The presence of positive social interaction effects has important policy implications. Positive social interaction effects reinforce behavioral changes produced by financial incentives, and, in addition, allow to reach a subgroup of the population not targeted by the social policy. First, this implies that to increase preventive behavior in the entire population, policymakers do not necessarily need to target everyone and can focus on subgroups as long as targeted and untargeted individuals interact with each other. Second, if positive spillovers are important, temporary subsidies can increase participation in behavior, and once a high adoption equilibrium is reached, it might be sustained even when subsidies are later scaled back. When, for example, imitation effects are driving the positive spillovers, participation becomes more attractive for an individual since utility from conforming to his or her peer's behavior increases as more social contacts participate. The rise in utility from imitation might compensate the utility decrease once subsidies are scaled back. Both points are important to allocate scarce public resources to attain health improvements. As Barham (2005), Gertler (2000, 2004) and Skoufias (2005) have shown, PROGRESA led to health improvements for children and adults and is a potential gamechanger in the human capital accumulation of children and households. The results we have obtained in this research project on policy effects and social interactions are specific to the studied setting. However, this settings is relevant for other countries as well. While Mexico was one of the first countries to set up a conditional cash transfer social program, a large number of Latin American and Asian countries have created similar programs.

Appendix 1: Construction of dependent variables

Table 2.10: Construction of dependent variables

	Survey March 1998 (pre-implementation)	Survey October 1998 (post-implementation)	Survey March 1999 (post-implementation)
Individual level data			
Annual growth and weight monitoring (yes=1; no=0)	at least 1 check-up in the past year	at least 1 check-up in the past 6 months (in each survey), recoded to at least 1 check-up in the past year	
Growth and weight monitoring at PROGRESA frequency (yes=1; no=0)	complies with the prescribed number of check-ups in the past year	not used	complies with the prescribed number of check-ups in the past 6 months
Vaccination against measles and tuberculosis (yes=1; no=0)	complies with age specific prescription for vaccination (children aged 5 or less)	complies with age specific prescription for vaccination (children aged 5 or less)	complies with age specific prescription for vaccination (children aged 2 or less)
Household (HH) level data			
Deworming drugs usage (yes=1; no=0)	someone in the HH treated in the past year	someone in the HH treated in the past 6 months (in each survey), recoded to at least someone treated in the past year	
Blood sugar test (yes=1; no=0)	someone in the HH treated in the past year	someone in the HH treated in the past 6 months (in each survey), recoded to at least someone treated in the past year	
Blood pressure test (yes=1; no=0)	someone in the HH treated in the past year	someone in the HH treated in the past 6 months (in each survey), recoded to at least someone treated in the past year	
Cervical screening (yes=1; no=0)	someone in the HH complies with screening norm in the past year	someone in the HH complies with screening norm in the past 6 months (in each survey), recoded to at least someone complies in the past year	

Individual level data are available on prevention use of children aged 5 years or younger. Prior to program initiation (March 1998 survey), it was asked whether a child had attended a growth and weight check-up in the past year and if so, how many times. After program implementation, the same questions were asked, but for the past six months (October 1998 and March 1999 survey). Two participation variables are constructed: one variable that indicates whether a child had attended at least one check-up in the past year (evaluated in March 1998 and March 1999), and another variable that indicates whether a child had attended the required number of growth and weight check-ups as imposed by PROGRESA, evaluated for the past year in March 1998 and for the past six months

in March 1999. For the latter participation variable, we choose to focus on the post-implementation period October 1998 to March 1999, rather than the period April 1998 to March 1999, since PROGRESA was only introduced in April 1998 and it is likely that a switch in monitoring frequency takes at least some transition time. Vaccination data are available on vaccination of measles, tuberculosis, tetanus and polio. In March 1998 and October 1998, vaccination history is recorded for children aged 5 or younger, while in March 1999, the information is available only for children aged 2 or younger. We focus on take-up of the vaccinations of tuberculosis and measles, since these are infrequent and therefore easily observed. There is one shot at birth for tuberculosis and one shot before age 1 for measles with a renewal around age 6. For tetanus and polio, there are at least four shots before the age of 5 and the data are not recorded accurately enough to follow the vaccination history unambiguously (Barham, 2005). When possible, we evaluate vaccination status in March 1999 and compare it with vaccination status in March 1998, however, for older children who are unobserved in March 1999, we derive post-program vaccination from the October 1998 survey.

For the usage of deworming drugs and the check-ups for blood sugar and blood pressure, household level data are available on whether or not someone in the household has taken these drugs or tests in the past year (March 1998 survey) or in the past six months (October 1998 and March 1999 survey). In the latter case, a yearly equivalent take-up variable is generated in order to analyze changes in yearly participation before and after program implementation. With respect to cervical screening, the data are also at the household level, but more information is available. In March 1998, participants were asked if someone in the household had ever participated in screening and if so, in which year. After program implementation, participants were asked whether someone in the household took a screening test in the last six months. In 1997, the official Mexican norm for cervical screening prescribed a test every three years (after normal test results for two consecutive years).[22] We create a variable that checks compliance with this norm both before (evaluated in March 1998) and after the implementation of PROGRESA (evaluated in March 1999) and analyze the changes in compliance.

Appendix 2: Descriptive statistics and attrition

[22]The recommended screening frequency is laid down by the official Mexican screening norm NOM-014-SSA2-1994 and its modifications.

Table 2.11: Descriptive statistics for the entire sample

	Eligible				Ineligible			
	Program	Control	Difference	(SD)	Program	Control	Difference	(SD)
HH: age	42.240	42.606	–0.366	(0.421)	51.504	51.661	–0.157	(0.563)
HH: literate	0.665	0.664	0.001	(0.023)	0.681	0.721	–0.040	(0.017)**
HH: female	0.083	0.086	–0.003	(0.006)	0.140	0.139	0.000	(0.009)
HH: speaks only indigenous language	0.050	0.058	–0.008	(0.014)	0.026	0.024	0.002	(0.008)
HH: speaks spanish and indigenous	0.373	0.380	–0.007	(0.049)	0.239	0.210	0.030	(0.041)
HH: degree in primary school	0.625	0.622	0.002	(0.021)	0.578	0.623	–0.450	(0.019)**
HH: degree in secondary school or beyond	0.053	0.050	0.003	(0.007)	0.089	0.083	0.006	(0.009)
HH: married	0.659	0.619	0.040	(0.023)*	0.620	0.621	–0.001	(0.020)
HH: partner but not married	0.215	0.259	–0.043	(0.021)**	0.146	0.160	–0.014	(0.016)
HH: seperated or alone	0.021	0.019	0.002	(0.003)	0.036	0.033	0.003	(0.004)
HH: widow	0.079	0.084	–0.005	(0.006)	0.141	0.132	0.009	(0.009)
P: age	31.293	31.423	–0.130	(0.421)	33.739	34.856	–1.117	(0.646)*
P: literate	0.516	0.507	0.009	(0.025)	0.472	0.498	–0.026	(0.017)
P: speaks only indigenous language	0.081	0.111	–0.030	(0.025)	0.037	0.039	–0.002	(0.011)
P: speaks spanish and indigenous	0.278	0.256	0.022	(0.037)	0.157	0.132	0.025	(0.029)
P: degree in primary school	0.507	0.497	0.011	(0.023)	0.414	0.452	–0.038	(0.016)**
P: degree in secundary school or beyond	0.033	0.043	–0.011	(0.005)**	0.068	0.062	0.006	(0.007)
household size	3.599	3.616	–0.017	(0.031)	2.904	2.965	–0.062	(0.046)
wealth: roof of tin	0.276	0.249	0.027	(0.028)	0.292	0.307	–0.015	(0.032)
wealth: roof of cement	0.074	0.082	–0.008	(0.013)	0.214	0.211	0.003	(0.024)
wealth: roof of tiles	0.115	0.084	0.031	(0.023)	0.127	0.098	0.029	(0.022)
wealth: floor of cement	0.253	0.220	0.033	(0.024)	0.547	0.555	–0.008	(0.029)
wealth: owner of agricultural land or animals	0.603	0.569	0.034	(0.029)	0.673	0.658	0.017	(0.022)
wealth: marginality index	639.346	638.767	0.579	(4.672)	841.460	847.290	–5.829	(8.131)

Note: HH refers to household head; P refers to partner. Mean values are reported of characteristics as measured in October 1997. Differences are estimated using OLS regression with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of differences: * p<0.10, ** p<0.05, *** p<0.01

Source: PROGRESA evaluation data

Table 2.12: Descriptive statistics for the sample of deworming drugs usage

	Eligible				Ineligible			
	Program	Control	Difference	(SD)	Program	Control	Difference	(SD)
HH: age	40.651	40.919	-0.268	(0.416)	49.965	49.979	-0.014	(0.619)
HH: literate	0.697	0.698	-0.001	(0.022)	0.705	0.741	-0.037	(0.018)**
HH: female	0.070	0.073	-0.002	(0.006)	0.129	0.133	-0.003	(0.009)
HH: speaks only indigenouse language	0.042	0.046	-0.004	(0.013)	0.023	0.021	0.002	(0.008)
HH: speaks spanish and indigenous	0.369	0.379	-0.011	(0.050)	0.232	0.204	0.028	(0.040)
HH: degree in primary school	0.655	0.652	0.003	(0.020)	0.595	0.643	-0.048	(0.019)**
HH: degree in secundary school or beyond	0.056	0.054	0.002	(0.007)	0.098	0.089	0.009	(0.010)
HH: married	0.681	0.637	0.045	(0.024)*	0.644	0.650	-0.006	(0.020)
HH: partner but not married	0.223	0.269	-0.046	(0.022)**	0.159	0.169	-0.010	(0.017)
HH: seperated or alone	0.018	0.017	0.002	(0.003)	0.031	0.030	0.001	(0.004)
HH: widow	0.056	0.063	-0.007	(0.005)	0.119	0.109	0.010	(0.009)
P: age	31.369	31.479	-0.110	(0.422)	34.166	35.336	-1.169	(0.660)*
P: literate	0.559	0.551	0.007	(0.026)	0.518	0.549	-0.031	(0.019)
P: speaks only indigenous language	0.074	0.103	-0.029	(0.024)	0.035	0.034	0.001	(0.011)
P: speaks spanish and indigenous	0.289	0.267	0.022	(0.039)	0.160	0.138	0.022	(0.030)
P: degree in primary school	0.548	0.537	0.011	(0.023)	0.451	0.496	-0.045	(0.017)**
P: degree in secundary school or beyond	0.036	0.048	-0.012	(0.006)**	0.078	0.069	0.009	(0.007)
household size	3.614	3.622	-0.008	(0.031)	2.876	2.962	-0.086	(0.043)**
wealth: roof of tin	0.280	0.250	0.030	(0.028)	0.298	0.315	-0.017	(0.032)
wealth: roof of cement	0.075	0.088	-0.012	(0.014)	0.211	0.212	-0.001	(0.024)
wealth: roof of tiles	0.114	0.076	0.038	(0.022)*	0.121	0.089	0.032	(0.021)
wealth: floor of cement	0.262	0.226	0.036	(0.025)	0.540	0.557	-0.017	(0.030)
wealth: owner of agricultural land or animals	0.597	0.562	0.035	(0.029)	0.663	0.652	0.011	(0.024)
wealth: marginality index	637.200	636.743	0.458	(4.811)	838.519	845.621	-7.101	(8.175)

Note: AW refers to answering woman. The values can best be compared to the values of partner of the entire sample. Mean values are reported of characteristics as measured in October 1997. Differences are estimated using OLS regression with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of differences: * p<0.10, ** p<0.05, *** p<0.01

Source: PROGRESA evaluation data

Table 2.13: Descriptive statistics for the sample of cervical cancer screening

	Eligible				Ineligible			
	Program	Control	Difference	(SD)	Program	Control	Difference	(SD)
HH: age	40.414	40.715	-0.301	(0.417)	49.539	49.620	-0.081	(0.620)
HH: literate	0.701	0.703	-0.002	(0.022)	0.712	0.746	-0.034	(0.018)*
HH: female	0.071	0.072	-0.001	(0.006)	0.135	0.138	-0.003	(0.010)
HH: speaks only indigenouse language	0.043	0.044	-0.001	(0.013)	0.023	0.022	0.001	(0.008)
HH: speaks spanish and indigenous	0.369	0.379	-0.009	(0.050)	0.236	0.201	0.034	(0.040)
HH: degree in primary school	0.660	0.657	0.003	(0.020)	0.600	0.648	-0.048	(0.019)**
HH: degree in secundary school or beyond	0.056	0.054	0.001	(0.007)	0.101	0.089	0.012	(0.010)
HH: married	0.688	0.645	0.043	(0.024)*	0.669	0.676	-0.007	(0.020)
HH: partner but not married	0.225	0.271	-0.046	(0.022)**	0.164	0.168	-0.004	(0.017)
HH: seperated or alone	0.017	0.014	0.003	(0.003)	0.024	0.026	-0.002	(0.004)
HH: widow	0.051	0.058	-0.007	(0.005)	0.107	0.101	0.006	(0.008)
P: age	31.727	31.816	-0.089	(0.417)	35.427	36.301	-0.874	(0.646)
P: literate	0.564	0.561	0.003	(0.026)	0.542	0.566	-0.025	(0.019)
P: speaks only indigenous language	0.075	0.101	-0.026	(0.024)	0.035	0.035	0.000	(0.011)
P: speaks spanish and indigenous	0.293	0.271	0.021	(0.040)	0.167	0.141	0.026	(0.031)
P: degree in primary school	0.555	0.548	0.007	(0.023)	0.471	0.510	-0.039	(0.018)**
P: degree in secundary school or beyond	0.036	0.048	-0.012	(0.006)**	0.082	0.073	0.008	(0.008)
household size	3.644	3.643	0.001	(0.030)	2.945	3.006	-0.061	(0.042)
wealth: roof of tin	0.281	0.252	0.029	(0.028)	0.293	0.312	-0.019	(0.031)
wealth: roof of cement	0.075	0.088	-0.013	(0.015)	0.223	0.214	0.009	(0.025)
wealth: roof of tiles	0.115	0.074	0.041	(0.022)*	0.120	0.090	0.030	(0.021)
wealth: floor of cement	0.264	0.225	0.039	(0.025)	0.554	0.562	-0.008	(0.030)
wealth: owner of agricultural land or animals	0.599	0.566	0.032	(0.029)	0.660	0.649	0.011	(0.024)
wealth: marginality index	636.561	636.764	-0.203	(4.899)	838.957	845.719	-6.762	(8.234)

Note: HH refers to household head; P refers to partner. Mean values are reported of characteristics as measured in October 1997. Differences are estimated using OLS regression with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of differences: * p<0.10, ** p<0.05, *** p<0.01

Source: PROGRESA evaluation data

Table 2.14: Descriptive statistics for the sample of blood sugar test

	Eligible				Ineligible			
	Program	Control	Difference	(SD)	Program	Control	Difference	(SD)
HH: age	40.803	41.190	-0.387	(0.430)	50.410	50.460	-0.050	(0.621)
HH: literate	0.697	0.696	0.000	(0.022)	0.700	0.736	-0.036	(0.018)**
HH: female	0.070	0.072	-0.003	(0.006)	0.133	0.135	-0.002	(0.010)
HH: speaks only indigenouse language	0.043	0.047	-0.004	(0.013)	0.023	0.022	0.001	(0.008)
HH: speaks spanish and indigenous	0.372	0.383	-0.010	(0.050)	0.231	0.204	0.028	(0.040)
HH: degree in primary school	0.656	0.652	0.004	(0.020)	0.595	0.643	-0.048	(0.019)**
HH: degree in secundary school or beyond	0.055	0.052	0.002	(0.007)	0.093	0.084	0.009	(0.010)
HH: married	0.680	0.637	0.044	(0.024)*	0.642	0.652	-0.011	(0.020)
HH: partner but not married	0.224	0.270	-0.046	(0.022)**	0.156	0.166	-0.009	(0.017)
HH: seperated or alone	0.018	0.015	0.003	(0.003)	0.031	0.030	0.001	(0.004)
HH: widow	0.056	0.064	-0.008	(0.006)	0.122	0.111	0.010	(0.009)
P: age	31.516	31.704	-0.188	(0.433)	34.270	35.605	-1.335	(0.665)**
P: literate	0.557	0.550	0.007	(0.026)	0.511	0.543	-0.032	(0.019)*
P: speaks only indigenous language	0.074	0.104	-0.030	(0.024)	0.035	0.035	0.000	(0.011)
P: speaks spanish and indigenous	0.292	0.272	0.020	(0.040)	0.159	0.139	0.020	(0.030)
P: degree in primary school	0.548	0.538	0.010	(0.023)	0.447	0.491	-0.044	(0.018)**
P: degree in secundary school or beyond	0.035	0.046	-0.011	(0.006)*	0.074	0.067	0.007	(0.007)
household size	3.618	3.617	0.002	(0.031)	2.858	2.952	-0.095	(0.044)**
wealth: roof of tin	0.279	0.250	0.029	(0.028)	0.298	0.316	-0.019	(0.031)
wealth: roof of cement	0.074	0.086	-0.013	(0.014)	0.207	0.210	-0.002	(0.024)
wealth: roof of tiles	0.115	0.075	0.040	(0.022)*	0.124	0.086	0.037	(0.021)*
wealth: floor of cement	0.260	0.224	0.036	(0.025)	0.539	0.555	-0.015	(0.030)
wealth: owner of agricultural land or animals	0.600	0.570	0.031	(0.029)	0.665	0.655	0.010	(0.024)
wealth: marginality index	637.199	637.484	-0.285	(4.856)	838.386	846.478	-8.092	(8.247)

Note: HH refers to household head; P refers to partner. Mean values are reported of characteristics as measured in October 1997. Differences are estimated using OLS regression with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of differences: * p<0.10, ** p<0.05, *** p<0.01

Source: PROGRESA evaluation data

Table 2.15: Descriptive statistics for the sample of blood pressure test

	Eligible				Ineligible			
	Program	Control	Difference	(SD)	Program	Control	Difference	(SD)
HH: age	40.794	41.157	-0.363	(0.428)	50.383	50.328	0.055	(0.616)
HH: literate	0.697	0.698	-0.001	(0.022)	0.700	0.739	-0.039	(0.018)**
HH: female	0.071	0.072	-0.001	(0.006)	0.133	0.136	-0.002	(0.010)
HH: speaks only indigenouse language	0.042	0.046	-0.003	(0.012)	0.024	0.021	0.003	(0.008)
HH: speaks spanish and indigenous	0.371	0.382	-0.011	(0.050)	0.232	0.203	0.029	(0.040)
HH: degree in primary school	0.656	0.654	0.002	(0.020)	0.594	0.644	-0.050	(0.019)***
HH: degree in secundary school or beyond	0.055	0.052	0.003	(0.007)	0.094	0.086	0.009	(0.010)
HH: married	0.681	0.637	0.044	(0.024)*	0.640	0.652	-0.012	(0.020)
HH: partner but not married	0.223	0.270	-0.047	(0.022)**	0.157	0.164	-0.008	(0.016)
HH: seperated or alone	0.019	0.016	0.003	(0.003)	0.032	0.030	0.001	(0.004)
HH: widow	0.056	0.064	-0.007	(0.006)	0.122	0.111	0.011	(0.009)
P: age	31.455	31.657	-0.202	(0.434)	34.200	35.479	-1.279	(0.668)*
P: literate	0.556	0.551	0.005	(0.025)	0.510	0.543	-0.033	(0.019)*
P: speaks only indigenous language	0.074	0.103	-0.029	(0.024)	0.035	0.034	0.001	(0.011)
P: speaks spanish and indigenous	0.290	0.270	0.020	(0.040)	0.159	0.136	0.022	(0.030)
P: degree in primary school	0.547	0.539	0.007	(0.023)	0.445	0.490	-0.045	(0.017)**
P: degree in secundary school or beyond	0.035	0.045	-0.010	(0.006)*	0.076	0.068	0.008	(0.007)
household size	3.618	3.617	0.001	(0.031)	2.855	2.941	-0.086	(0.044)*
wealth: roof of tin	0.279	0.252	0.027	(0.028)	0.296	0.317	-0.020	(0.032)
wealth: roof of cement	0.075	0.086	-0.011	(0.014)	0.210	0.209	0.002	(0.024)
wealth: roof of tiles	0.115	0.075	0.039	(0.022)*	0.122	0.090	0.031	(0.021)
wealth: floor of cement	0.262	0.225	0.037	(0.025)	0.538	0.555	-0.017	(0.030)
wealth: owner of agricultural land or animals	0.600	0.570	0.030	(0.029)	0.663	0.652	0.011	(0.024)
wealth: marginality index	637.236	637.127	0.109	(4.865)	838.681	846.336	-7.655	(8.153)

Note: HH refers to household head; P refers to partner. Mean values are reported of characteristics as measured in October 1997. Differences are estimated using OLS regression with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of differences: * p<0.10, ** p<0.05, *** p<0.01

Source: PROGRESA evaluation data

Table 2.16: Descriptive statistics for the sample of growth and weight monitoring of children aged 5 years or younger

	Eligible				Ineligible			
	Program	Control	Difference	(SD)	Program	Control	Difference	(SD)
HH: age	37.028	37.574	-0.546	(0.477)	42.727	43.430	-0.703	(0.803)
HH: literate	0.750	0.735	0.015	(0.021)	0.808	0.833	-0.025	(0.019)
HH: female	0.044	0.051	-0.006	(0.006)	0.072	0.066	0.006	(0.011)
HH: speaks only indigenouse language	0.033	0.038	-0.005	(0.011)	0.018	0.022	-0.004	(0.010)
HH: speaks spanish and indigenous	0.382	0.384	-0.002	(0.054)	0.229	0.199	0.030	(0.047)
HH: degree in primary school	0.698	0.678	0.020	(0.020)	0.657	0.683	-0.026	(0.026)
HH: degree in secundary school or beyond	0.072	0.070	0.002	(0.010)	0.157	0.150	0.007	(0.018)
HH: married	0.698	0.639	0.058	(0.029)**	0.704	0.708	-0.004	(0.031)
HH: partner but not married	0.238	0.299	-0.061	(0.028)**	0.186	0.199	-0.013	(0.028)
HH: seperated or alone	0.012	0.009	0.002	(0.003)	0.018	0.017	0.000	(0.005)
HH: widow	0.039	0.046	-0.007	(0.005)	0.074	0.059	0.015	(0.012)
P: age	29.313	29.749	-0.435	(0.484)	32.203	33.988	-1.784	(0.861)**
P: literate	0.611	0.610	0.001	(0.026)	0.652	0.686	-0.034	(0.025)
P: speaks only indigenous language	0.074	0.092	-0.018	(0.023)	0.043	0.047	-0.004	(0.018)
P: speaks spanish and indigenous	0.307	0.282	0.025	(0.044)	0.169	0.143	0.027	(0.036)
P: degree in primary school	0.596	0.588	0.008	(0.024)	0.526	0.584	-0.059	(0.025)**
P: degree in secundary school or beyond	0.045	0.063	-0.018	(0.008)**	0.147	0.115	0.032	(0.015)**
household size	3.938	3.971	-0.033	(0.038)	3.585	3.750	-0.165	(0.066)**
wealth: roof of tin	0.276	0.256	0.020	(0.030)	0.277	0.317	-0.040	(0.037)
wealth: roof of cement	0.085	0.090	-0.005	(0.016)	0.290	0.286	0.004	(0.034)
wealth: roof of tiles	0.106	0.072	0.034	(0.022)	0.084	0.059	0.026	(0.019)
wealth: floor of cement	0.280	0.241	0.039	(0.027)	0.625	0.652	-0.026	(0.035)
wealth: owner of agricultural land or animals	0.577	0.542	0.035	(0.033)	0.633	0.636	-0.003	(0.033)
wealth: marginality index	616.705	616.172	0.533	(5.733)	827.859	834.459	-6.600	(8.612)

Note: HH refers to household head; P refers to partner. Mean values are reported of characteristics as measured in October 1997. Differences are estimated using OLS regression with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of differences: * p<0.10, ** p<0.05, *** p<0.01

Source: PROGRESA evaluation data

Table 2.17: Descriptive statistics for the sample of vaccination of children aged 5 years or younger

	Eligible				Ineligible			
	Program	Control	Difference	(SD)	Program	Control	Difference	(SD)
HH: age	37.252	37.757	-0.505	(0.441)	43.374	43.535	-0.161	(0.806)
HH: literate	0.743	0.732	0.011	(0.022)	0.803	0.827	-0.024	(0.019)
HH: female	0.046	0.053	-0.007	(0.006)	0.073	0.067	0.006	(0.011)
HH: speaks only indigenouse language	0.032	0.039	-0.007	(0.012)	0.020	0.022	-0.001	(0.010)
HH: speaks spanish and indigenous	0.380	0.374	0.006	(0.053)	0.243	0.195	0.048	(0.048)
HH: degree in primary school	0.690	0.681	0.009	(0.021)	0.658	0.682	-0.023	(0.026)
HH: degree in secundary school or beyond	0.074	0.068	0.006	(0.010)	0.153	0.144	0.008	(0.018)
HH: married	0.692	0.653	0.039	(0.029)	0.707	0.693	0.014	(0.032)
HH: partner but not married	0.241	0.285	-0.044	(0.028)	0.185	0.210	-0.024	(0.029)
HH: seperated or alone	0.012	0.011	0.001	(0.003)	0.018	0.019	0.000	(0.005)
HH: widow	0.041	0.045	-0.004	(0.005)	0.070	0.061	0.009	(0.011)
P: age	29.460	29.956	-0.496	(0.480)	32.884	33.909	-1.025	(0.861)
P: literate	0.607	0.613	-0.006	(0.027)	0.645	0.677	-0.033	(0.026)
P: speaks only indigenous language	0.074	0.094	-0.020	(0.024)	0.040	0.042	-0.001	(0.017)
P: speaks spanish and indigenous	0.306	0.274	0.032	(0.044)	0.185	0.143	0.042	(0.037)
P: degree in primary school	0.590	0.588	0.002	(0.025)	0.529	0.570	-0.041	(0.025)
P: degree in secundary school or beyond	0.047	0.065	-0.018	(0.009)**	0.132	0.115	0.017	(0.015)
household size	3.949	3.966	-0.017	(0.037)	3.633	3.751	-0.118	(0.068)*
wealth: roof of tin	0.265	0.249	0.016	(0.030)	0.258	0.309	-0.051	(0.037)
wealth: roof of cement	0.086	0.089	-0.003	(0.016)	0.295	0.288	0.006	(0.034)
wealth: roof of tiles	0.109	0.084	0.025	(0.023)	0.085	0.063	0.022	(0.021)
wealth: floor of cement	0.279	0.241	0.037	(0.027)	0.607	0.648	-0.041	(0.034)
wealth: owner of agricultural land or animals	0.585	0.542	0.043	(0.033)	0.637	0.648	-0.011	(0.032)
wealth: marginality index	616.048	615.021	1.027	(5.831)	828.488	832.185	-3.697	(8.388)

Note: HH refers to household head; P refers to partner. Mean values are reported of characteristics as measured in October 1997. Differences are estimated using OLS regression with robust standard errors that allow for correlation of disturbance terms within localities. Significance levels of differences: * p<0.10, ** p<0.05, *** p<0.01

Source: PROGRESA evaluation data

Table 2.18: Relation between attrition and household characteristics

	All households		**Eligible households**		**Non-eligible households**	
Treatment village	0.002	(0.015)	-0.013	(0.016)	0.017	(0.017)
Very high village marginality index (ref. high)	-0.021	(0.018)	-0.012	(0.018)	-0.023	(0.020)
Fraction eligible individuals in village	-0.030	(0.038)	-0.013	(0.038)	-0.077	(0.048)
Household marginality index	0.000	(0.000)	0.000	(0.000)	0.000	(0.000)
HH: primary education	-0.023	(0.011)*	-0.021	(0.016)	-0.024	(0.013)*
HH: degree in secundary education or beyond	0.005	(0.017)	-0.007	(0.024)	0.016	(0.022)
HH: educational information missing	-0.021	(0.027)	-0.083	(0.030)***	0.030	(0.042)
HH: literate	-0.008	(0.011)	-0.014	(0.013)	-0.002	(0.015)
HH: age	-0.002	(0.000)***	-0.002	(0.000)***	-0.001	(0.000)***
HH: speaks indigenous language	0.014	(0.017)	0.004	(0.015)	0.019	(0.023)
HH: language information missing	-0.018	(0.045)	-0.058	(0.073)	0.000	(0.057)
P: primary	-0.024	(0.012)**	-0.042	(0.015)***	-0.004	(0.017)
P: secundary	-0.016	(0.016)	-0.040	(0.024)*	0.007	(0.021)
P: literate	-0.003	(0.011)	0.017	(0.015)	-0.023	(0.016)
P: age	0.000	(0.000)	0.000	(0.000)	0.000	(0.000)
P: speaks indigenous language	-0.023	(0.012)*	-0.031	(0.014)**	-0.007	(0.016)
Civil status						
Married	0.003	(0.020)	0.010	(0.033)	0.002	(0.027)
Partner but not married	-0.003	(0.020)	-0.002	(0.033)	0.003	(0.028)
Separated or alone	0.030	(0.022)	0.057	(0.040)	0.014	(0.027)
Widow	0.000	(0.018)	0.016	(0.032)	-0.008	(0.022)
Household members (ref. 1 member)						
2 household members	-0.078	(0.016)***	-0.074	(0.035)**	-0.084	(0.018)***
3 household members	-0.117	(0.017)***	-0.108	(0.036)***	-0.130	(0.019)***
4 household members	-0.134	(0.018)***	-0.156	(0.039)***	-0.129	(0.021)***
5 household members	-0.138	(0.018)***	-0.151	(0.040)***	-0.145	(0.020)***
6 or more household members	-0.143	(0.019)***	-0.165	(0.040)***	-0.139	(0.022)***
1 child	0.000	(0.009)	-0.007	(0.019)	0.000	(0.010)
2 children	0.003	(0.012)	0.004	(0.023)	0.001	(0.014)
3 children	-0.011	(0.014)	-0.007	(0.024)	-0.021	(0.017)
4 children	-0.011	(0.015)	-0.011	(0.026)	-0.008	(0.018)
5 children	-0.011	(0.018)	-0.014	(0.027)	0.004	(0.025)
6 or more children	-0.009	(0.018)	-0.014	(0.028)	0.009	(0.028)
Wealth indicators						
Floor: earth	-0.013	(0.019)	0.003	(0.023)	-0.028	(0.026)
Floor: cement	-0.008	(0.019)	0.000	(0.024)	-0.015	(0.025)
Roof: Asbestos	0.005	(0.021)	-0.007	(0.030)	0.023	(0.024)
Roof: Tin	-0.035	(0.016)**	-0.037	(0.021)*	-0.025	(0.022)
Roof: Carton	-0.008	(0.019)	-0.004	(0.024)	-0.014	(0.025)
Roof: Palm leaves	-0.040	(0.015)**	-0.045	(0.019)	-0.031	(0.022)
Roof: Tiles	0.006	(0.025)	-0.003	(0.024)	0.024	(0.036)
Roof: cement blocks	0.022	(0.019)	-0.004	(0.024)	0.037	(0.025)
Owner of agricultural land	-0.021	(0.011)*	-0.018	(0.014)	-0.025	(0.011)**
Constant	0.417	(0.049)***	0.487	(0.073)***	0.396	(0.065)***

Note: HH refers to household head; P refers to partner. Attrition is estimated using OLS regression with robust standard errors that allow for correlation of disturbance terms within localities. Standard errors between brackets. Significance levels of coefficients: * p<0.10, ** p<0.05, *** p<0.01
Source: PROGRESA evaluation data

Chapter 3

Unintended spillover effects of influenza vaccination: a regression discontinuity approach.

Joint work with Anne Gielen and Tom Van Ourti

3.1 Introduction

We study spillover effects of preventive care policy on those individuals not targeted by the policy. Many countries have preventive care policies in place that target specific groups. For example, secondary preventive care – such as screening for breast, colon or cervical cancer –, but also primary prevention for infectious diseases – such as vaccination for influenza – is mostly recommended and/or incentivized for well-defined (often age-based) groups in society. Target groups are mostly derived from observational studies describing who is most at risk of contracting the disease, in combination with randomized controlled trials (RCT) that analyze whether the policies/interventions – think of screening, vaccinations, etc. – do have beneficial effects on health outcomes. The findings, while useful, provide an incomplete picture by overlooking spillovers on the preventive behavior of those not targeted; and the studies that do consider spillovers tend to suffer from endogeneity biases or limited external validity.[1] Moreover, RCTs are mostly silent on effects among targeted and non-targeted individuals on – arguably equally important – outcomes such

[1] For infectious diseases, there is the additional concern that the potentially affected preventive behavior of the non-targeted population might affect disease prevalence among the targeted population (see e.g. Ward (2014)). Our study accommodates such externality effects, but does not estimate the magnitude of these externalities.

as medical care savings or sickness absence. Both types of neglected effects do affect costs and benefits of preventive care policies and therefore constitute essential information to guide optimal policy design and targeting of these policies.

Our particular application analyzes the influenza vaccination policy in the Netherlands. Since 1996/1997, all Dutchmen aged 65 years and over (or suffering from certain specific diseases) receive a personal invitation for a free flu shot. This allows to quantify the behavioral change in terms of vaccination take-up, health outcomes and medical care consumption among individuals crossing the age threshold. While the effects on the targeted population are useful for policy design, our primary aim is to estimate policy induced spillovers onto non-targeted individuals which has proven difficult in earlier work (e.g. Rao et al., 2012). We use the subpopulation of children of the targeted over-65 population since information on family ties in Dutch administrative records help us overcome the usual identification challenges and allows the estimation of spillover effects from targeted parents to their non-targeted adult children in terms of sickness absence, medical care consumption, health outcomes and vaccination behavior. The 'adult children' in our data are between 20 and 51 and generally do not cohabit with their parents.

In the Netherlands, it is estimated that about 10% of the population is affected by influenza.[2] Around 160 GP consultations and 12 hospitalizations per 10,000 individuals have been estimated to be influenza-related. Age standardized mortality of influenza (as primary cause) is around 0.075 per 10,000 individuals, but influenza is also related to cardiovascular and pneumonia mortality, leading to an overall age-standardized influenza-related mortality rate of about 1 per 10,000 individuals of which 95% among individuals aged 60 or more. Comparison of incidence rates of influenza and influenza related medical care use and mortality across countries is difficult due to differences in surveillance methods, and definitions of influenza (Meerhof et al., 2004), but a common feature across temperate regions of the world is that influenza tends to recur every year.[3] Annual vaccination is the leading worldwide strategy for reducing the public health burden of influenza. The Netherlands adopted a protective vaccination policy targeted at chronically ill individuals with specific disorders, with the intention to directly protect individuals who are most at risk of influenza mortality. To increase participation, the Dutch health authorities initiated in 1991 a series of interventions, tailored to the eligible population, including

[2] Obtaining exact estimates of the health burden of influenza is difficult due to the bias introduced by vaccination coverage.

[3] The exact timing, duration and gravity of the flu season differs beween countries and from year to year (WHO, 2014; CDC, 2014).

public information campaigns, systematic documentation of individuals at risk, and monitoring of vaccination participation. A major reform in 1996 extended the vaccination target group by adding all healthy individuals aged 65 and over to the eligible population. In addition, barriers to take up vaccination were reduced for the eligible population by offering free vaccination, sending personalized invitation letters, and simplifying the process to obtain a free vaccination, but also by incentivizing the supply side by providing remuneration to health care providers in charge of the vaccination program.[4] The new policies were successful and vaccination rates in the Dutch target group increased rapidly after 1996 (van Essen et al., 2001; CBS, 2003; Mereckiene et al., 2012, 2014). Currently, in line with guidelines of the European Commission, most European countries recommend free influenza vaccination for the elderly and the chronically ill (European Commission, 2014).[5] Recommendations for flu shots also include younger age groups in the United States, but health insurance plans might not cover the costs of flu shots.[6]

The crucial identifying element in our study design is the quasi-random variation introduced by the personal invitations for a free influenza vaccination for individuals aged 65 and over since 1996. We exploit the resulting discontinuity at the 65-age threshold with a fuzzy regression discontinuity (RD) design. The RD design is applied on survey data and administrative records from Statistics Netherlands for the identification of the policy-induced behavioral responses among the targeted population and spillover effects among their adult children, under fairly weak assumptions. Age-triggered (sharp and fuzzy) RD designs have been applied in other contexts (Battestin et al., 2008; Card et al., 2008-2009; Lemieux and Milligan, 2008; Carpenter and Dobkin, 2009; Stancanelli and Van Soest, 2012), but is particularly attractive in the case of the Dutch influenza vaccination policy given that not actual age, but age on May 1st determines whether one receives an invitation for a free flu shot, and thus removes the typical concern that the treatment of interest (invitation for a free flu shot) coincides with eligibility for other welfare programs or government transfers such as retirement benefits which are based on actual age.[7]

Our results indicate that the vaccination policies targeted to the elderly increase immunization by almost 20 percentage points (from about 30% to 50%) at age 65. This direct

[4] In 2008, the age threshold was lowered to 60, but this policy change is not analyzed in this paper.

[5] In only 4 EU member state countries, the full cost of the vaccination is borne by the recipient (O'Flanagan et al., 2012).

[6] Since 2010, CDC recommends influenza vaccination for everyone older than 6 months (CDC, 2010), and since the affordable care act flu shots are included in most health insurance plans.

[7] In the Netherlands, the health insurance system was dramatically reformed in 2006. However, neither before 2006, and neither after 2006, there has been a discontinuity in the coverage of medical care insurance (in general and for flu shots) at the age of 65 (Mossialos and Thomson, 2002; Roos and Schut, 2008).

effect is accompanied by a decrease in the vaccination behavior of their non-targeted adult children from around 9 to 5 percent. These direct and spillover effects are strong and have an impact over and beyond the vaccination decisions of the (un)targeted individuals. For example, we estimate that the influenza vaccination program saves 0.8 individuals out of 100,000 at the age threshold. We also find a decline of around 10 percentage points at age 65 in the number of individuals that consult a general practitioner (GP) (from around 50% to 40%) and the consumption of prescribed medicines (from around 70% to 60%) during the typical influenza months, but not during other months. Mortality and GP visits of their adult children are not affected, but the occurrence of flu-like symptoms increases from 45% to 55% and sickness absence among this group increases with 8 percentage points (from 14% to 22%), suggesting that the lack of mortality and GP effects among adult children is due to their lower age, and not due to their relatively smaller change in vaccination behavior (as compared to that of the elderly parents). For both parents and adult children, we find no statistically significant effects on hospital admissions, visits to medical specialists, and consumption of unprescribed medicines. All our findings are robust to different specifications at the 65-age threshold, and robustness checks suggests that the identifying assumptions of our fuzzy RD design hold.

Interpretation of the direct policy effects is straightforward as the reduced mortality, GP visits and prescribed medicines at age 65 can be safely ascribed to the vaccination policy at this age threshold. The estimates of primary interest – the (unintended) spillover effects among the adult children of the targeted population – are more difficult to interpret. We explore several possible channels that might have generated the negative spillover effect and find suggestive evidence that a social stigma cost is revealed to the adult children when their oldest parent crosses the age threshold. A potential trigger for the social stigma cost is the explicit framing of the target group in the invitation letter sent out to eligible parents.

Our research fits into the literature on the benefits of influenza vaccination, but also in the broader literature that studies (unintended) spillover effects (and social interactions) on individuals not targeted by a policy. In the influenza domain, few studies analyze spillover effects of influenza vaccination (e.g. Jordan et al., 2006; Thomas et al., 2006), and none of these studies considers the behavioral response in terms of vaccination uptake of the non-targeted group. They rather evaluate mortality and morbidity outcomes which might not only be affected by changes in the vaccination behavior of the non-targeted group, but also by changes in herd immunity. Existing studies also suffer from inconclusive evidence due to limitations in the study design, i.e. observational studies that

are prone to the typical biases of selection, omitted variables bias and reverse causality; and RCT's that might be based on selected samples or have only limited external validity (Nichol, 2008). Our study improves upon the existing evidence by providing causal evidence of the spillover effects on the adult children of the targeted population, by exploiting the random variation created by a population-wide vaccination program in the Netherlands. Our study is also related to Ward (2014) which is, as far as we know, the first study of externality effects. Ward (2014) evaluates the expansion of coverage from the traditional target population (the elderly and the chronically ill) to the entire population (including children and adults) in Ontario, Canada. She finds substantial decreases in influenza-related mortality and respiratory hospital admissions among the traditional target population. Her study is explicitly concerned with herd immunity in the sense that she studies the effect of increased take-up among the newly targeted population (the children and adults) on mortality, morbidity and medical care use of the traditional target population, keeping the vaccination behavior of the latter fixed. Our study focuses instead on spillover effects that are triggered by changes in the vaccination behavior of the non-targeted group (in our case: adult children), and our age-triggered RD design makes sure that we condition on the overall vaccination coverage in the Netherlands.[8] Our study shows that such (unintended) spillover effects can be substantial, and have adverse consequences on morbidity and mortality of the untargeted population, which is in line with the efficacy and effectiveness of influenza vaccinations being higher among younger compared to older individuals (Grohskopf et al., 2013).

Our work is also broadly related to the literature on social interactions (see Dahl et al. (2014) for a recent example). A social interaction effect occurs when an individual's participation decision relates to the participation decision of others in an individual's social group. Our RD design – in combination with exogenous social groups (in our case: families) – is perfectly suited to address the typical identification challenges of omitted variables bias, reverse causality and endogenous group membership that arise when estimating social interaction effects.[9] Kremer & Miguel (2007) argue that social interactions might arise from (1) a desire to imitate the decisions of one's social contacts, (2) sharing information or perceptions about costs and benefits of using the technology or medication[10]; or stigma costs which might arise in our application on free influenza vaccination

[8] Individuals with parents just below and above the age threshold face the same herd immunity or overall vaccination coverage.

[9] One might worry that social interactions within the family are biased by shared family genes and habits, but this would only be the case in our setup when unobserved family effects differ at both sides of the age threshold.

[10] Costs and benefits in this context should be interpreted broadly. Research, within the context of

since perceptions about the target group (individuals with specific disorders and the elderly) may matter for the participation of untargeted individuals (Moffitt, 1983)[11], or (3) from a reduced personal incentive to vaccinate due to (epidemiological) externalities.[12] We provide suggestive evidence that social stigma effects are the dominant channel for the observed negative spillover effects.

While our estimates of the positive direct policy effects and the negative spillover effects are based on population-wide random variation and have therefore higher external validity compared to RCT which tend to be based on non-representative samples of the population, one should remain prudent in extrapolating our estimates since they are local around the 65-age threshold. This limitation is particularly binding for the spillover effects, but less for the direct policy effects since efficacy and effectiveness of influenza vaccinations is higher among younger compared to older individuals (Grohskopf et al., 2013). One should also be careful when interpreting our spillover effects since all estimates in our study reflect 'intentions to treat' (ITT). Since we do not observe the vaccination of the targeted parents and their untargeted children in the same dataset, we directly link the age of the parents to the behavior of their children.

At the same time, our findings are useful for policy. Our results suggest that vaccination policy in the Netherlands is successful in increasing vaccination coverage among targeted individuals and leads to substantial reductions in mortality, GP visits and the use of prescribed medicines, but also generates negative spillover effects between targeted and untargeted individuals and increases influenza-related symptoms and sickness absence with respectively 10 and 8 percentage points among the latter group. The Dutch vaccination programs should pay more attention to the effects of information dissemination on public perceptions and attitudes on (voluntary) vaccination. One possible policy adjustment in this respect could be to less explicitly focus on the delineation of the target group, or alternatively, more explicitly focus on the benefits of indirect protection (next to direct protection) when addressing the target group and the broader population. Our

the Dutch influenza vaccination program and in other settings, shows that besides tangible costs, other elements play a role in vaccination take-up, such as perceptions about the effectiveness and the safety of the vaccine, beliefs about the infection risk and disease complications, fear about side effects, needles and pain, knowledge on insurance coverage and time costs (Bish et al., 2011; Chapman et al., 1999; Nagata et al., 2011; Rao et al., 2012; van Essen et al., 1997; Wu, 2003; Zijtregtop et al., 2010).

[11] A study by Krooneman & Verheij (2003) among 4000 Dutch citizens shows for example that the most cited reason by healthy adults not to get vaccinated is that they do not belong to the target group (reported by 56% of the healthy adults).

[12] Miguel and Kremer (2007) also mention sharing information on how to use a certain technology, but since influenza vaccination is administered by a general practitioner or a medical assistant, this channel is less relevant in our setting.

findings also suggests that many (European) countries with similar influenza vaccination policies in place might face similar negative spillover effects amongst family members.

The remainder of chapter 3 is structured as follows. Section 3.2 presents background information on the Dutch influenza vaccination program, discusses the empirical approach and explains the identification strategy. In section 3.3, we describe the data and section 3.4 discusses the main results. In section 3.5, we look for the underlying channels that might drive the spillover effects. Section 3.6 concludes.

3.2 Identification

3.2.1 Identification challenges

Identifying spillover effects within groups has proven to be difficult since one faces the important identification challenges of reverse causality, omitted variables bias (or correlated unobservables) and endogenous group membership (or selection bias) (Manski, 1993; Moffitt, 2001). Reverse causality arises if one person's decisions affect another person's decisions and vice versa. This generates an identification problem when one attempts to simultaneously regress both person's behavior on the other person's behavior. The omitted variables bias problem surfaces when not all relevant characteristics of individuals involved in the spillovers within the group are controlled for. The problem of endogenous group membership implies that individuals tend to engage in social contacts with people who have similar tastes, attitudes and preferences. This might drive the correlation in behavior among people that have frequent social contacts, and one might mistakingly interpret this correlation as a spillover effect.

We overcome the identification problems of reverse causality bias and omitted variables bias by considering a random subset of individuals (in our setting parents) in a social group (families) that are offered program participation at a different price (free influenza vaccination and invitation letter). This exogenous variation allows analyzing how others (adult children) in the group change their behavior. Endogenous group membership is addressed by studying spillover effects within families. Since children do not choose their parents, selection issues do not matter when analyzing spillover effects.[13,14] We provide

[13] More generally, seasonal influenza infections and vaccination decisions are rather unlikely to change existing social connections or create new ones.

[14] The selection issue might be seen as a special case of the omitted variables bias problem and shows that the choice of families is not essential for our identification strategy (it is however essential to choose a group within which spillover effects are studied). Within this interpretation, family characteristics or

more details of our empirical models in section 3.2.5.

3.2.2 The design of the Dutch free influenza vaccination program

3.2.2.1 Dutch influenza vaccinations before 1996

In the Netherlands, increased influenza activity is typically recorded between mid-November and early March, and flu shots are usually delivered between October and December. In order to reduce influenza related hospital admissions, morbidity and mortality during this period, the Dutch Health council and health authorities identify high-risk groups who are targeted for influenza vaccination. In the eighties high-risk groups were defined based exclusively on existing chronic disorders, such as diabetes, cardiovascular and pulmonary conditions, HIV/AIDS, renal disease and immune dysfunctions. High-risk individuals who were insured by a regional sickness fund obtained vaccination free of charge.[15] High-risk individuals who were covered by a private insurer or individuals who did not belong to the high-risk group had to pay to receive vaccination.[16] The take-up rate in the high-risk group was rather constant and remained below 30% (Fedson et al., 1995; van Essen et al., 2001).

In 1991, the national health authorities concluded that vaccination coverage among chronically ill and elderly individuals was inadequate. A series of interventions were started to increase participation. First, the general public and at risk patients were informed about the existence and the benefits of influenza vaccination. Second, the position of the general practitioner (GP) – who occupies the role of gatekeeper in the Dutch health care system – was strengthened. The GP was encouraged to register and personally invite at risk patients and organize clearly communicated vaccination moments.[17] The central role of the GP and the increased publicity rapidly increased influenza vaccination in the high-risk group from about 30% in 1991 to 50% in 1995 (see Figure 3.1).

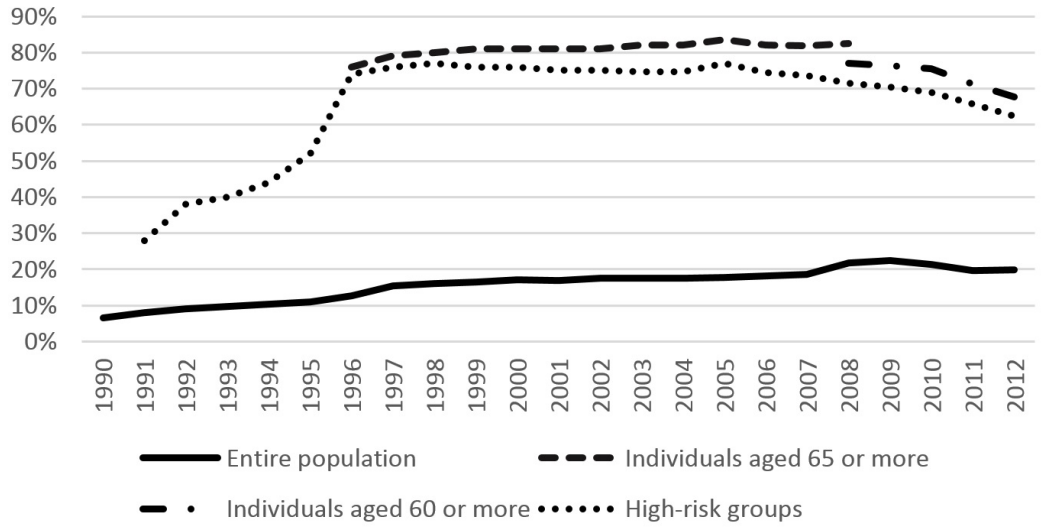
(health) endowments that are correlated within families and that remain unobserved, will be addressed by the RD identification strategy which assumes that the unobserved family characteristics or (health endowments) in close vicinity to the discontinuity are randomly distributed.

[15] In the eighties, nineties and up to 2005, two thirds of the population - whose earnings (employment or replacement income) fell below an income threshold - were compulsory insured for health care by a regional sickness fund. The remaining third could (voluntarily) enrol in private insurance.

[16] More specifically, in 1996 an individual had to pay 58.20 guilders to be vaccinated against influenza, which corresponds to 37.78 euros (in 2013 purchasing power).

[17] The fraction of GPs that effectively heed these additional tasks increased rapidly. Registration of at risk patients in a computer program was performed by 54% of the GP in 1994 and 82% in 1997. Personal invitations were sent out by 40% in 1994, 77% in 1997 and 95% in 2000. Special vaccination hours were organized by 72% in 1994, 86% in 1997 and 90% in 2000 (Hak et al., 2000; Tacken et al., 2002).

Figure 3.1: Vaccination rates in the Netherlands



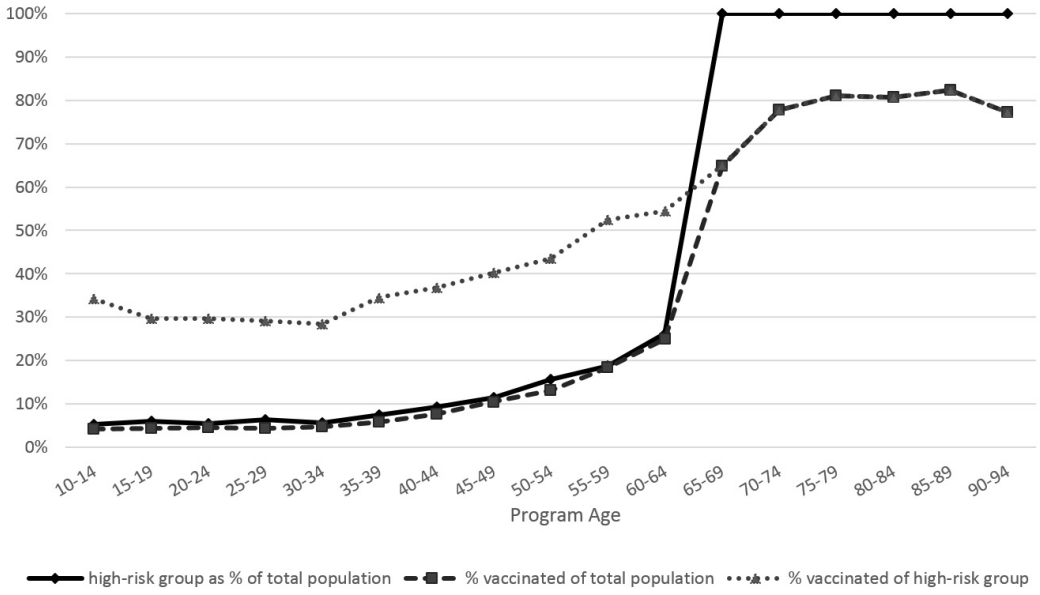
Source: Van Essen et al. (2001) ; CBS (2003) ; de Bakker (2001); Tacken et al. (2002-2010); Jansen (2013)

3.2.2.2 The Dutch influenza vaccination program since 1996

In 1996, a major reform was rolled out. All (healthy) individuals aged 65 or above were added to the high-risk group. In addition, all high-risk individuals were made eligible for free influenza vaccination[18], received a personalized invitation (around late September/early October) and GPs directly received a remuneration per vaccinated individual of the high-risk population. At the same time new supporting agencies were set up and influenza prevention evolved into a nationwide preventive care program. The provision of influenza vaccinations to the high-risk group was simplified. Prior to the reform, an individual first needed to get a prescription from the GP, next go to the pharmacist to buy the vaccine and revisit the GP for the administration of the vaccine. After the reform, every GP had their own stock of vaccines which were directly administered to eligible individuals. However, for individuals not targeted by the vaccination program, the old arrangement still applied.

[18]Note that some employers provide (free or subsidized) flu shots to their employees. In the period 1997-1998 to 2007-2008, 14% of the individuals who were vaccinated and who did not belong to the high-risk group, indicated to do this at the initiative of their employer (own computations, health interview surveys).

Figure 3.2: Vaccination rates and high-risk group fraction by program age for the (pooled) influenza seasons 1997-1998 up to 2007-2008.



Source: Health Interview Survey, own computations.

As can be seen in Figure 3.1, the reform led to a sharp increase in vaccination rates of the at risk population in 1996/1997 and further increased until 2005 both in the entire population (up to 19%) and among individuals aged 65 or above (up to 80%).[19] The vaccination rate among high-risk individuals is one of the highest in Europe (Mereckiene et al., 2012, 2014). Since 2006, participation in the high-risk group based on chronic illnesses, is slightly decreasing.

By 1998, the high-risk group covered 19.3% of the Dutch population, of whom 12.7% above and 6.6% below the age threshold (Tacken et al., 2003). Figure 3.2 shows the fraction of individuals that belong to the high-risk group decomposed by age. The receipt of a personal invitation and a free flu shot at age 65, led to a sharp increase of vaccination take-up of individuals at the age threshold. Participation rates climb further up to around age 70 and stabilize thereafter. Carman & Mosca (2011) provide evidence that, within the context of the Dutch influenza program, individuals above 65 who start vaccinating against influenza continue to do so in the next year.

[19]Note that the overall vaccination rate of 19% is well below the rate where marginal benefits of additional vaccination coverage tend to zero. Using Canadian data of 1996-2006 – on a similar vaccination program as that in place in the Netherlands, that was expanded to the full population – , Ward (2014) estimates that the point of zero marginal benefits is reached when the overall vaccination rate is around 33%

3.2.3 Validity of the age-based RD design

RD designs based on discontinuities in age have the advantage that age cannot be manipulated, but are therefore different from most other RD designs since everyone eventually crosses the age threshold. Lee and Lemieux (2010) mention three potential concerns. First, "one must consider the possibility that the age of interest is causing eligibility for potentially many other programs, which could affect the outcome" (ibidem, cit, p. 346). Second, the effect on the outcome must be immediate; and lastly, as individuals approach the age threshold, they should not anticipate program eligibility (i.e. crossing the age threshold) and change their behavior. The second and third concern should be small since influenza vaccination is protective after about 2 weeks, and needs to be taken yearly to guarantee effectiveness, implying that anticipating or postponing behavior is not useful. The age eligibility criteria for the free influenza vaccination program in the Netherlands counteracts the first concern.

The program eligible age (being over-65) is computed on May 1st, at the end of an influenza season, and determines whether an individual will receive an invitation in September/October of the preceding year. For example, for the influenza season of 1998-1999, eligibility for the program is evaluated based on whether one will be 65 years or over on May 1st, 1999; and determines whether an individual receives a personal invitation in September/October 1998 at the start of the influenza season and a free flu shot between October and December 1998. Therefore, each year, more or less 7 out of 12 of the newly invited individuals are 64 years old at the start of the influenza season.[20] This feature of the vaccination program removes the concern that eligibility for other benefits that start on the day one turns 65 in the Netherlands (e.g. pension claims, benefits for the elderly), interferes with eligibility for influenza vaccination as the start of eligibility only coincides for those that turn 65 on May 1st. In section 3.4.4 we provide additional checks of the internal validity of our RD design.

3.2.4 Relation of the identification strategy to existing research

We exploit an age discontinuity to learn about spillover effects from parents to their adult children in the context of influenza vaccinations. Our identification strategy is reminiscent to other studies that exploit age discontinuities in different contexts, including

[20] For example, for the influenza season 1998-1999, all individuals that turn 65 between May 2nd 1998 and May 1st 1999 are newly invited. Hence, all those that turn 65 between May 2nd 1998 and the day they receive the invitation for the influenza vaccination (late September/early October) are 65, while all other newly invited are 64 when they receive the invitation.

moral hazard effects of social assistance (Lemieux and Milligan, 2008), the retirement consumption puzzle (Battestin et al., 2008), retirement and home production (Stancanelli and Van Soest, 2012), health care use (Card et al., 2008), and mortality (Card et al., 2009; Carpenter and Dobkin, 2009).

At the same time, we must also overcome the typical challenges faced when studying spillover effects and social interactions (see also section 3.2.1). Early research estimated social interaction effects by linking individual propensity to behave in a particular way to the average behavior in a social group, mostly defined by the researcher based on ethnicity or geographic proximity. It has been criticized for not overcoming biases due to reverse causality, omitted variables and endogenous group membership.[21] Others have used information on existing social groups rather than defining a potential social network. More precise information provides better prospects to control for individual and group characteristics and reduces bias due to correlated unobservables[22], but the approach cannot generally address selection issues (Cohen-Cole & Fletcher, 2008b).[23] More recent identification strategies rely on random variation to address self-selection and other identification threats. Randomized group assignment has been used by Carrell et al. (2011) who analyze fitness outcomes among students at the US Air Force academy who are randomly assigned to squadrons. Other examples use first year college students who are randomly assigned a roommate and may influence each other's lifestyle choices (e.g. Duncan et al., 2005; Yakusheva et al., 2011; Kremer & Levy (2008). While much can be learned from its design, the problem with this type of research is that social groups are sometimes created artificially and it is difficult to establish whether estimates are specific to the created situation or capture adequately social interaction effects present in the real world. Estimates obtained from randomized treatment assignment in naturally occurring social groups are therefore potentially more convincing. This approach has been successfully ap-

[21] The inclusion of controls (individual and group-level characteristics and fixed effects) can reduce the biases, but cannot generally guarantee these biases are satisfactorily addressed. Important examples of this early approach in the health domain are Aizer & Currie (2004) who analyse social interactions in publicly funded prenatal care and delivery services use within ethnic groups; and Deri (2005) who presents evidence of peer effects in health service utilization in Canada. There is also a related approach that studies social multipliers, which eliminates the concerns for reverse causality, but not necessarily the other endogeneity concerns. Health-related applications of this approach are Auld (2011) on social interaction effects in body weight, Gray (2013) for breast cancer prevention and Apouey & Picone (2014) for malaria preventive behaviour.

[22] Panel data is often used to overcome reverse causality.

[23] There are several examples in the health domain. Christakis and Fowler (2007, 2008) study obesity and smoking, and Rosenquist et al. (2010) study alcohol consumption. Several studies used Add Health Data which is particularly suited to analyze how social contexts affect adolescents' health and risk-taking behaviors (e.g. Trogdon et al., 2008; Cohen-Cole & Fletcher, 2008a; Halliday & Kwak, 2009; Fletcher, 2010-2012; Card & Giuliano, 2013).

plied in developing countries. Kremer & Miguel (2007) analyze social interaction effects in the usage of deworming drugs in Kenya using information on household social links and by exploiting a random assignment of schools to the intervention. Oster & Thornton (2012) look at the role of social interactions in the usage of menstrual cups in Nepal in a school environment, and Bouckaert (2014) studies the participation in preventive care exploiting the introduction of the PROGRESA social welfare program in Mexico. Rao et al. (2012) is a rare example where social interactions in health care usage are estimated in the developed world. They study vaccination decisions among US students by using random variation in the ease with which students have access to vaccination locations. Methodologically, the approach of Dahl et al. (2014) is closest to our work in that it combines an RD design for randomized treatment assignment in combination with an exogenously determined social group. The authors exploit a discontinuity in the Norwegian parental leave system to estimate peer effects in parental leave take-up within workplace and family networks.

3.2.5 Empirical models

We exploit an age-triggered discontinuity in the costs of influenza vaccination among individuals aged 65 or above and analyze the influence on vaccination rates among their adult children : all individuals above the age threshold are personally invited by their GP to receive influenza vaccination free of charge, while individuals below the age threshold have to pay and put in more effort to receive influenza vaccination. The main idea is that the treatment of parents, the invitation letter and the free vaccination, might influence vaccination behavior of their adult children

$$V_i^c = \alpha^c + \beta^c V_i^p + \varepsilon_i^c \tag{3.1}$$

where vaccination participation V_i^c of child i is related to their parent's vaccination behavior V_i^p and unobservable variables ε_i^c.[24] In our fuzzy RD setting, the participation behavior of the parent can be written as:

$$V_i^p = \alpha^p + g^p(age_i^p - t) + h^p(age_i^p - t)T_i^p + \gamma^p T_i^p + \varepsilon_i^p \tag{3.2}$$

[24] For notational simplicity, we do not include observable characteristics.

where the age threshold is denoted by t, T_i^p is a treatment dummy that equals one if $age_i^p \geq t$, and $g^p(.)$ and $h^p(.)$ are unknown functional forms.[25] The jump in parents' vaccination rates at the threshold is captured by γ^p which measures the divergence in behavior between first-year-treated individuals and nearly treated individuals. It represents the combined effect of the invitation letter and the free flu shot on the vaccination behavior of the parents. Equation (3.1)-(3.2) show that reverse causality is broken since T_i^p is included in the parent's equation, but not in the child's equation. Plugging equation (3.2) into equation (3.1) gives the reduced form of equation (3.1) and relates the treatment of the parent T_i^p directly to the child's take-up of flu shots:

$$V_i^c = \alpha^c + \beta^c \left[\alpha^p + g^p(age_i^p - t) + h^p(age_i^p - t)T_i^p + \gamma^p T_i^p + \varepsilon_i^p\right] + \varepsilon_i^c \tag{3.3}$$

Equation (3.3) reveals that omitted variables bias is eliminated since treatment T_i^p is not correlated with unobserved parent and child characteristics as treatment is quasi-random.[26] Equation (3.3) can be further simplified to:

$$V_i^c = \delta^c + g^c(age_i^p - t) + h^c(age_i^p - t)T_i^p + \lambda^c T_i^p + \upsilon_i^c \tag{3.4}$$

where λ^c is the treatment spillover of the parent's treatment on the child's vaccinating behavior. We use equations (3.2) and (3.4) in the empirical part, and not equation (3.1), since we do not observe the vaccination take-up of parents and children in the same dataset (see section 3.3). It follows that our estimates provide 'intentions to treat' (ITT).[27]

We also analyze the effect of the vaccination policy at age 65 on two additional sets of outcomes: (1) parental mortality, morbidity and medical care use; and (2) mortality, morbidity, medical care use and sickness absence among their adult children. For example, in case of the direct policy effects on parental mortality, this can be represented by equation (3.2), the first stage, and equation (3.5) which shows how parental vaccination behavior impacts upon parental mortality (M_i^p):

[25] For small windows, a linear specification is a good approximation for $g^p(.)$ and $h^p(.)$, polynomials (or local linear regressions) are preferred for larger windows.

[26] For example, potential worries about bias due to shared family genes and habits do not apply in this setting.

[27] The 2SLS system defined by equations (3.1) and (3.2) is exactly identified with parent's treatment instrumenting for vaccination take-up. Hence, if information on parent's and child's influenza immunization were available in the same data, we would be able to compute the social interaction effect (β^c) as the ratio of the reduced form coefficient λ^c, and the first stage treatment coefficient γ^p (Lee & Lemieux, 2010). In absence of that information, Angrist and Krueger (1992) showed that a 2SLS regression can also be consistently estimated on two separate data sets (termed Two Sample IV (TSIV) or Two Sample 2SLS (TS2SLS)), under the premise that the two samples are drawn from the same underlying population.

$$M_i^p = \alpha^{po} + \beta^{po} V_i^p + \varepsilon_i^{po} \tag{3.5}$$

While quasi-random variation in invitations for free flu shots was sufficient to guarantee identification of equation (3.1), equation (3.5) requires the additional 2SLS assumption that parental treatment should only affect parental mortality via parental influenza vaccination. This seems an unrealistically strong assumption, since vaccination behavior of the parents might affect vaccination behavior of their children, and hence might affect the probability that the parents get influenza, and die from it.[28,29] The same problem occurs when one consider spillover effects among the children on the other outcomes. Also here we need to assume that parental vaccination does not directly influence the outcome of the child. For these reasons, we present ITT estimates for the effects on the other outcomes of the children and the parents. These ITT can be interpreted in a causal way but for the effect of vaccination behavior of both children and parents combined.

3.3 Data

3.3.1 Data Sources

Our analysis uses linked survey and administrative data from Statistics Netherlands that can be merged at the individual level using unique individual identifiers.

Our main data source is the annual cross-sectional health interview survey (HIS) 1997-2008, that is part of a more general household survey (POLS). The HIS samples constitute a representative cross-section of the non-institutionalized Dutch population.[30] A wave includes around 10,000 respondents.[31] We have extensive information on health, demographic and socioeconomic characteristics. The most crucial indicators for our purposes are the individual's date of birth and vaccination history. In the analysis that considers the impact of parental age on parental vaccination (and other outcomes), date of birth is

[28] A partial way out would be to estimate equation (3.5) on the subset of parents whose children do not vaccinate, and the subset of parents whose children do vaccinate. This is not feasible with our data.

[29] We mentioned in the introduction that our approach conditions on herd immunity (Ward, 2014). This is not in contrast with what we write here. Our identification strategy accounts for herd immunity since parents just below/above the age threshold face the same herd immunity. However, here we consider the possibility of within-family externalities and these are not addressed by our RD approach.

[30] A multistage cluster sampling design was used: First, a number of municipalities are selected within all regions of the Netherlands (probability of selection is related to the population size). Second, within each municipality, individuals are selected. The number of selected individuals depends on the size of the municipality with a minimum per municipality.

[31] More specifically: 10,898 in 1997, 9,323 in 1998, 9,877 in 1999, 9,922 in 2000, 9,676 in 2001, 9,745 in 2002, 9,876 in 2003, 11,117 in 2004, 10,378 in 2005, 9,607 in 2006, 8,741 in 2007, 9,499 in 2008.

used to construct program age in years, which is used as the running variable in our RD design. In the analysis on the spillover effects, we derive "age of the child" from date of birth and use it as a control variable. Vaccination history informs whether the individual ever had an influenza vaccination, and reports month and year of the last flu shot; but cannot be readily used since a typical influenza season does not coincide with the civil year.

A typical influenza season in the Netherlands ranges from September to May with an increased activity between November and March. This period does not coincide with the wave period, which follows the civil year.[32] Since the month in which the questionnaire was completed is known, we have rearranged the waves into influenza seasons that start in September and end in August in the following year. We created a dummy variable that equals one [zero] if the individual has [not] vaccinated against influenza during the influenza season. However, since most shots are reported to be taken in the period September to January[33], individuals interviewed in this period might report not having taken a flu shot, but might still vaccinate in the near future. For this particular group, the dummy equals one if they received an influenza vaccination in the past influenza season, since this is by far the best predictor for a renewed vaccine take-up (Carman & Mosca, 2011).[34]

Next to vaccination behavior, we also study the effects of the vaccination program on a wider set of outcome variables. The HIS informs on the occurrence (yes/no) of flu-like symptoms during the last two months. When the individual answers positively, the individual is asked whether these flu-like symptoms have led to sickness absence (recorded as a binary variable). Medical care use is summarized with two binary indicators: one for GP visits during the last two months, and another for visits to the medical specialist during the last two months. Medicine use is reported as a binary variable during the last

[32] Survey months are almost uniformly distributed. There is a slight underrepresentation of July and August, which are the main holiday months.

[33] More specifically, 95% of all reported influenza shots are reported to be taken in this period, and 86% in October and November, which is the recommended vaccination period. 4.5% in September, 2% in December and 2% in January.

[34] Not unexpectedly, this procedure has the biggest effect on individuals surveyed in the month September, but a much smaller effect in October-January. The imputation procedure might create some measurement error in our dependent variable and warrants scrutiny. For the interview months September to January, the vaccination dummy for the newly invited individuals (i.e. those that are 65 on May 1st) is partly based on their vaccination behavior in the previous year. Given that there is a discontinuity at 65, one might worry that our imputation leads to an underestimation of the jump. One way to verify this, is to analyze the discontinuity in vaccination behavior at 65 using only those individuals that were interviewed between February and August. Our estimates show that the age discontinuity in vaccination behavior of the parents is slightly larger (0.017 percentage points), but not statistically different from the one that is obtained from including the observations for September to January.

month, separately for prescribed and non-prescribed medicines. Each of these variables will feature as outcome variable for parents and adult children, except sickness absence which will only be used for the children.

The HIS also provides us with a set of control variables: gender, highest obtained educational degree (assembled in four categories), household composition (single person, couple, household with children, other), number of household members, sector of employment, existing medical conditions (in order to identify individuals who belong to the high-risk group based on chronic disorders), and presence of a chronic illness.

We obtain additional information from administrative data of Statistics Netherlands. These data are needed since we do not observe parental age of the individuals included in the HIS. We retrieve parental age of all individuals in the HIS from the municipal registries ("GBA"), which contain data on all residents of the Netherlands. First, a specific registry provides information that links children with parents. For almost 75% of our HIS sample, we observe the parent's personal identifier.[35] The parent identifiers can be used to obtain their date of birth[36], gender and nationality. When both parents are alive, we focus on the oldest parent to estimate equations (3.2) and (3.4). This guarantees that no other parent already qualifies for free influenza vaccination based on age-eligibility, and avoids that we need to define our running variable – program age – separately for fathers and mothers. Second, for each individual in the HIS and their oldest parent in GBA, we have an encrypted address location, which can be linked to a neighborhood – a small geographical unit within a municipality –, the population density in the neighborhood, the municipality and any larger geographical division. As vaccination invitation letters are sent out at the end of September, we fix the addresses for each influenza season on October 1st. The geographic coordinates of each municipality are known, which allows to calculate the distance between children's and parents' municipalities of residence.

The administrative data are also needed to study the mortality effects among the parents, and their adult children. The mortality registry ("DO") includes all deaths, the date of death, and the cause of death, based on the ICD-10 classification. This allows isolating influenza-related deaths. However, simply recording influenza-coded mortality would lead to an underestimation of the death burden of influenza, since mortality more

[35] We only observe parents if they were alive sometime between 1995 and 2010 and lived in the Netherlands. The individuals without observed parents are predominantly older individuals and individuals of foreign origin.

[36] Similar to the date of birth in the HIS, it is used to construct the parental program age variables, the running variable.

frequently results from complications attributable to influenza than from influenza itself. In general, there are two approaches in estimating influenza-associated mortality: (1) analyzing cause-coded death notifications due to pneumonia and influenza or even all respiratory, and circulatory conditions; (2) monitoring all-cause mortality (Nicoll et al., 2012). We construct four binary indicators reflecting whether someone died during the influenza season.[37] The narrowest definition considers influenza or pneumonia deaths. The second category considers diseases of the respiratory system, and the third category combines diseases of the respiratory and circulatory system. The last considers all-cause mortality.

3.3.2 Sample restrictions

Since the linked data are first recorded in 1997, the influenza season 1996-1997 is only partly observed, and hence the corresponding observations for 1997 in HIS are dropped. We pool all waves to provide sufficient statistical power to our fuzzy RD design. We use four combinations of the HIS and the administrative records in our estimations. Two consider HIS as the main source of information. The first ("parent HIS") does not exploit the linkage with the administrative records and is used to estimate the effect of the vaccination policy on vaccination behavior (, medical care use, and medicine consumption) at age 65. The second ("child HIS") does exploit the linkage to parental age in the administrative data and is used to estimate the spillovers of the policy on the behavior of the children. The two other datasets are based on the administrative data and are used to estimate mortality effects.[38] Since mortality (and particularly flu-related mortality) are rare events, we do not exploit the linkage with the HIS. This reduces the number of available controls[39], but provides maximum power as every Dutchman is included in the administrative records. The third dataset ("parent admin") is used to estimate mortality effects at age 65, and the fourth ("child admin") exploits the link with parental age to estimate spillover effects on child mortality.

"Parent HIS" restricts the pooled HIS cross sections to individuals (i) who have filled out the question related to vaccination take-up, i.e. 57% of the pooled cross sections[40],

[37] We consider someone dying of a specific cause when the primary *or* secondary cause of death equals the specific cause. The primary cause of death is the initial disease. Complications of the initial disease are secondary causes.

[38] In the mortality data, we also observe all deaths in 1996, and therefore analyze influenza seasons 1996/1997 to 2007/2008.

[39] Note that controls only serve to make RD designs more precise.

[40] There are three types of non-response: the questions with respect to influenza vaccination are only asked to individuals aged 12 years or above; they are surveyed in the written subpart of the HIS, which

Table 3.1: Descriptive statistics

Panel A: Descriptive statistics for Child HIS and Parent HIS

	Child HIS			Parent HIS	
	Full sample: Program age 20-51 (irrespective of oldest parent's age)	Sample: oldest parent's program age 63-66	Sample: oldest parent's program age 62-67	Sample: program age 63-66	Sample: program age 62-67
Sample Characteristics					
Vaccination rate	0.07 (0.25)	0.06 (0.24)	0.06 (0.24)	0.40 (0.49)	0.40 (0.49)
Vaccination rate (survey month between February and August)	0.06 (0.24)	0.06 (0.23)	0.05 (0.23)	0.41 (0.49)	0.41 (0.49)
Influenza-like symptoms past 2 months (0=no; 1=yes)	0.45 (0.50)	0.47 (0.50)	0.47 (0.50)	0.31 (0.46)	0.32 (0.47)
Non-prescribed medication past month (0=no; 1=yes)	0.49 (0.50)	0.50 (0.50)	0.50 (0.50)	0.40 (0.49)	0.40 (0.49)
Prescribed medication past month (0=no; 1=yes)	0.31 (0.46)	0.29 (0.45)	0.29 (0.45)	0.65 (0.48)	0.65 (0.48)
Visited GP past 2 months (0=no; 1=yes)	0.32 (0.46)	0.31 (0.46)	0.31 (0.46)	0.42 (0.49)	0.42 (0.49)
Visited medical specialist past 2 months (0=no; 1=yes)	0.14 (0.35)	0.13 (0.34)	0.13 (0.34)	0.23 (0.42)	0.23 (0.42)
Sickness absence past 2 months (0=no; 1=yes)	0.16 (0.37)	0.17 (0.38)	0.17 (0.38)		
Male (0=no; 1=yes)	0.49 (0.50)	0.49 (0.50)	0.49 (0.50)	0.51 (0.50)	0.50 (0.50)
Program age	37.61 (8.05)	35.33 (4.40)	35.24 (4.52)	64.42 (1.11)	64.34 (1.70)
Risk group (0=no; 1=yes)	0.08 (0.28)	0.07 (0.26)	0.07 (0.26)	0.25 (0.43)	0.25 (0.43)
Chronic illness (0=no; 1=yes)	0.27 (0.44)	0.26 (0.44)	0.26 (0.44)	0.47 (0.50)	0.47 (0.50)
Education level 1: primary (0=no; 1=yes)	0.08 (0.27)	0.06 (0.24)	0.06 (0.24)	0.23 (0.42)	0.23 (0.42)
Education level 2: lower secondary (0=no; 1=yes)	0.20 (0.40)	0.18 (0.38)	0.18 (0.38)	0.31 (0.46)	0.30 (0.46)
Education level 3: upper secondary (0=no; 1=yes)	0.42 (0.49)	0.44 (0.50)	0.44 (0.50)	0.27 (0.44)	0.28 (0.45)
Education level 4: post-secondary (0=no; 1=yes)	0.30 (0.46)	0.32 (0.47)	0.32 (0.47)	0.19 (0.40)	0.19 (0.39)
Number of household members	3.05 (1.32)	3.13 (1.29)	3.10 (1.30)	1.89 (0.57)	1.89 (0.57)
Family type: single person (0=no; 1=yes)	0.13 (0.34)	0.12 (0.33)	0.13 (0.34)	0.19 (0.40)	0.20 (0.40)
Family type: couple (0=no; 1=yes)	0.22 (0.42)	0.20 (0.40)	0.20 (0.40)	0.72 (0.45)	0.72 (0.45)
Family type: household with children (0=no; 1=yes)	0.63 (0.48)	0.67 (0.47)	0.66 (0.47)	0.07 (0.26)	0.08 (0.27)
Family type: other (0=no; 1=yes)	0.01 (0.11)	0.01 (0.08)	0.01 (0.08)	0.01 (0.10)	0.01 (0.10)
Population density: below 500 inhabitants/km^2 (0=no; 1=yes)	0.14 (0.35)	0.14 (0.35)	0.14 (0.35)	0.15 (0.36)	0.15 (0.36)
Population density: between 500 and 2500 inhabitants/km^2 (0=no; 1=yes)	0.68 (0.47)	0.68 (0.46)	0.68 (0.47)	0.70 (0.46)	0.70 (0.46)
Population density: above 2500 inhabitants/km^2 (0=no;1=yes)	0.18 (0.38)	0.18 (0.38)	0.18 (0.38)	0.15 (0.36)	0.15 (0.36)
Employment sector: health care provision (0=no; 1=yes)	0.13 (0.34)	0.13 (0.33)	0.13 (0.34)		
Oldest parent male (0=no; 1=yes)	0.57 (0.50)	0.61 (0.49)	0.62 (0.49)		
Oldest parent's program age	67.24 (9.81)	64.48 (1.11)	64.45 (1.72)		
Distance children – parents (in km)	24.03 (41.45)	23.91 (41.48)	24.41 (41.23)		
N	33,852	3,112	4,720	3,183	4,792

Note: we report the mean and standard deviation (in parentheses)

Panel B: Descriptive statistics for Child Admin and Parent Admin

	Child Admin			Parent Admin	
	Full sample: Program age 20-51 (irrespective of oldest parent's age)	Sample: oldest parent's program age 63-66	Sample: oldest parent's program age 62-67	Sample: program age 63-66	Sample: program age 62-67
Sample Characteristics					
Male (0=no; 1=yes)	0.51 (0.50)	0.51 (0.50)	0.51 (0.50)	0.50 (0.50)	0.50 (0.50)
Program age	34.74 (5.38)	34.57 (5.25)	34.49 (5.23)	64.46 (1.12)	64.41 (1.71)
Mortality: pneumonia and influenza (Dec-June)	0.00002 (0.006)	0.00002 (0.006)	0.00002 (0.006)	0.0005 (0.022)	0.0005 (0.022)
Mortality: respiratory diseases (Dec-June)	0.00004 (0.007)	0.00004 (0.007)	0.00004 (0.007)	0.0011 (0.033)	0.0011 (0.034)
Mortality: respiratory and circulatory diseases (Dec-June)	0.00013 (0.012)	0.00013 (0.012)	0.00013 (0.012)	0.0033 (0.058)	0.0034 (0.058)
Mortality: all-cause mortality (Dec-June)	0.00046 (0.028)	0.00045 (0.027)	0.00046 (0.028)	0.0070 (0.083)	0.0070 (0.084)
Oldest parent male (0=no; 1=yes)	0.71 (0.45)	0.72 (0.45)	0.71 (0.45)		
Oldest parent's program age	64.48 (2.89)	64.50 (1.12)	64.50 (1.32)		
N	23,330,790	9,320,945	14,009,238	7,163,278	10,779,851

Note: we report the mean and standard deviation (in parentheses)

and (ii) who fall within a specified window around the 65-age threshold. In "Child HIS", the windows apply to the oldest parent's program age. It additionally restricts to those individuals whose parents are alive and credibly identified from the administrative records[41], i.e. almost 75% of the remaining sample. "Parent admin" and "child admin" are derived from the pooled administrative records of all Dutchmen, and no additional data selection criteria are used, except that respectively own age and parental age should fall within the windows. For all analyses, we selected two windows: a window of +/- 2 years around the age threshold, based on the criterion by Imbens and Kalyanaraman (2012), and a window of +/- 3 years around the age threshold in order to increase the statistical power and precision in the estimation of vaccination behavior and health outcomes of adult children.[42]

Table 3.1 presents the descriptive statistics for the different datasets. The HIS samples are shown in panel A and the administrative datasets in panel B. We find very little, if any, difference between the narrow and broad window.

Columns 2 to 4 in panel A show the average characteristics of the adult children with parents whose age falls within the specified parental age windows as well as the average characteristics of all individuals between 20 and 51 in our sample (i.e. the observed minimum and maximum age when the parent's age is restricted to differ between 15 and 45 years with the child's age). Vaccination rates show that we study a subpopulation with a below average vaccination coverage (see also Figure 3.1). Only 6% of the individuals in our sample has been vaccinated during the current influenza season. Almost 50% has suffered from influenza-like symptoms in the past 2 months, resulting in sickness absence for 17% of the individuals. GP visits and usage of prescription medicines is around 30%. About half of the individuals in our restricted samples are male and about two thirds live in a household with children. A quarter of the sample suffers from a chronic illness and 7% belongs to the high-risk group, who qualifies for free influenza vaccination based on existing disorders. The oldest parent is more likely to be male and the parent's age profile is almost perfectly centred around the age threshold. Individuals tend to live close

has a lower response rate; and some individuals chose not to answer the question.

[41] If the difference between a child's and a parent's age falls below 15 years or exceeds 45 years, the observations are considered as outliers and excluded from the analysis. The restriction is binding for less than 0.5% of the individuals with parents.

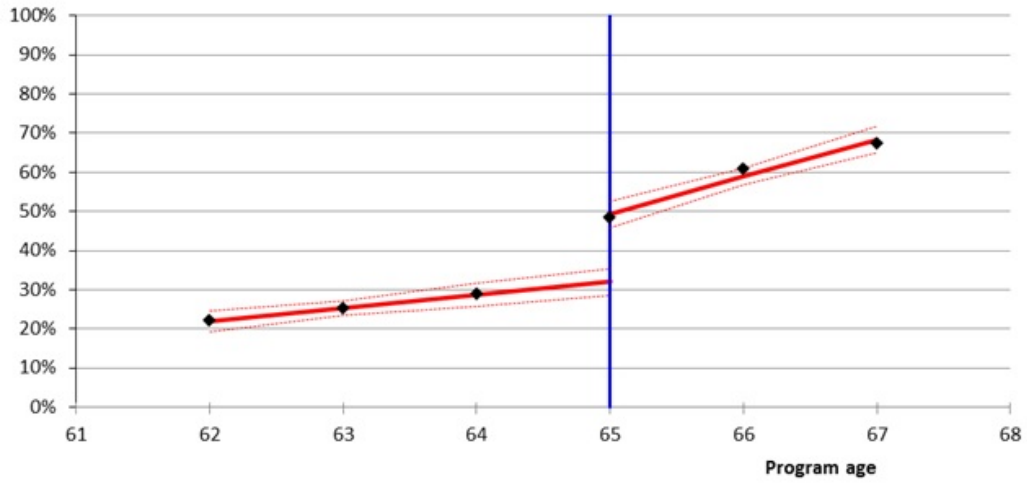
[42] Power analysis shows for example that we need more than 5000 observation to detect significant differences in average vaccination behavior of adult children at both sides of the cut-off with power 80% and type I error of 5%. Limiting the bandwidth to +/- 2 years around the age threshold provides a sample of 3112 individuals, increasing the bandwidth with 1 additional year at each side increases the sample to 4720 observations. The power of a test is the probability to correctly reject the null hypothesis when the null hypothesis is false.

to their parents, with on average 24 km distance between the municipalities of residence. In fact, 55% of our sample lives in the same municipality as their parents.

If we compare the characteristics of the individuals in the age window restricted samples and those in the general sample of individuals of the same age, we conclude that they are very similar, except perhaps for parent's age. This is again reassuring with respect to our sampling strategy.

Columns 5 and 6 in panel A show the average characteristics of the individuals in "parent HIS". Around 40% of the individuals around the 65-age threshold has been vaccinated during the current influenza season, and this rate is only marginally higher for those surveyed between February and August.[43] Around 30% of the individuals suffered from influenza-like symptoms in the past 2 months, a smaller fraction than the one in the Child HIS. Consultations of the GP and the medical specialists, on the other hand, are more frequent with visit rates of 42% and 23%, respectively. Moreover, about two thirds of the elderly has used prescription medicines in the past month. About three quarters of the individuals live in a couple, and only 7% has cohabiting children. About half of the sample suffers from a chronic illness and one quarter belongs to the high-risk group, who qualifies for free influenza vaccination based on existing disorders.

Figure 3.3: Influenza vaccination rate of individuals around the 65-age threshold



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate, grouped in yearly bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

[43] See also footnote 34.

Panel B in Table 3.1 provides average characteristics of the administrative datasets used to analyze mortality effects. Both the child and parent sample are balanced with respect to gender. We consider mortality between December and June (see below). Amongst adult children, mortality rates are 2, 4, 13 and 45 deaths per 100,000 individuals, respectively for pneumonia and influenza, respiratory diseases, respiratory and circulatory diseases combined and all-cause mortality. Amongst individuals around the age threshold, mortality rates are higher, respectively at 5, 11, 33 and 70 deaths per 100,000 individuals.

Table 3.2: RD estimates of policy effects at the 65-age threshold

	+/- 2 years window around the age threshold	**+/- 3 years window around the age threshold**
Policy effect (γ^p)	0.168***	0.184***
Standard error	(0.035)	(0.026)
N	3183	4792

Notes: See text and Section 3.2 for details on the RD set-up. Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. We use linear trends in program age that can differ at each side of the cutoff. Control variables include dummies for gender, member of risk group based on existing disorders, population density, chronic illness, education level, number of household members, family type, influenza season (see also Table 3.1). OLS regression estimates are reported that use sample weights and cluster standard errors at the wave-municipality level to mimic the sample design. Significance level: * p<0.10, ** p<0.05, *** p<0.01.

3.4 Results: Direct and spillover effects of the Dutch influenza vaccination policy

3.4.1 Direct policy effect: vaccination at age 65

A major advantage of an RD design is that the magnitude of the effects can easily be visualized using graphical methods. Figure 3.2 suggest an age discontinuity in vaccination take-up at age 65, but this is more clearly visible from Figure 3.3 which shows influenza vaccination take-up in a +/- 3 year window surrounding the 65-age threshold. Yearly bins are used (see also section 3.3.1). The Appendix provides additional graphs with a larger window, smaller bins and quadratic trends (Figures 3.5 to 3.7).

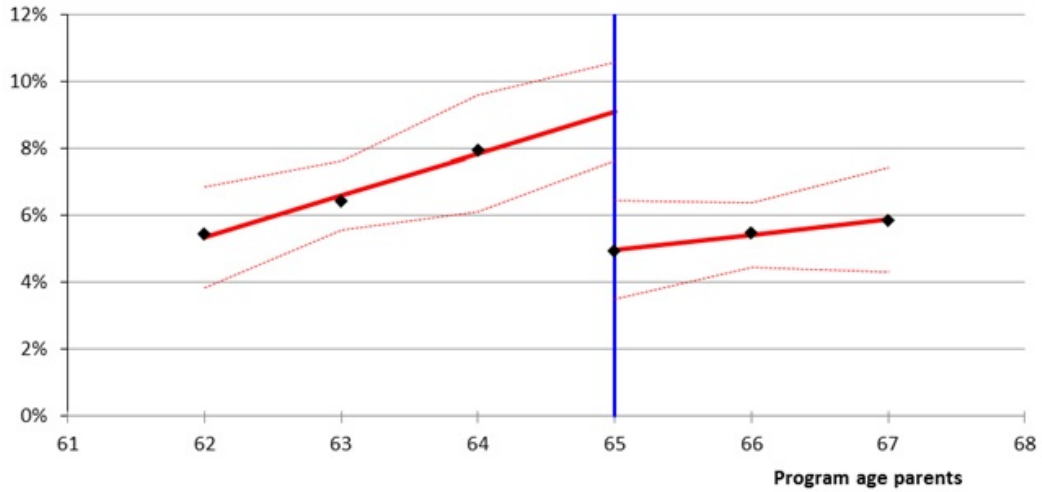
Figure 3.3 reveals a clear discontinuity at the age threshold of about 18 percentage points in the vaccination rate. This is an important policy effect implying a direct increase in take-up at the threshold from about 30% to 50% after receiving a personal invitation for a free influenza vaccination. There is an increasing trend on both sides of the threshold, but the increase in vaccination rates is steeper among age-eligible individuals. Carman &

Mosca (2011) suggest that this is due to a further influx into the immunization program and a negligible drop-out rate. Table 3.2 shows regression estimates of the magnitude of the policy effect at age 65, denoted γ^p in equation (3.2).[44] The estimates indicate a similar, and highly significant, increase in influenza vaccination rates for the +/- 3 year window. The smaller window leads to a slightly lower point estimate. It is reassuring that the jump in the RD graph does not change after adding a large set of control variables.

3.4.2 Spillover effect: vaccination among adult children

Having established the presence of a policy effect in the previous subsection, we now turn to the spillover effects on the vaccination behavior of the adult children which are presented in Figure 3.4. Take-up rates to the right [left] of the age threshold are from adult children whose oldest parent does [not] qualify for free influenza vaccination based on age. A negative discontinuity of around 4 percentage points at the cutoff is observed. This is an important decline in relative terms from around 9% to 5%. Not only do we observe a jump at the age threshold, also the increasing slope in vaccination uptake before the age threshold has diminished. The Appendix provides additional graphs with a larger window, smaller bins and quadratic trends (Figures 3.8 to 3.10).

Figure 3.4: Influenza vaccination rate of children



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate of adult children, grouped in yearly bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

[44] Marginal effects obtained from a probit specification reveal slightly larger point estimates: 0.183 [0.197] for the +/- 2 [3] year window.

Table 3.3: RD estimates for spillover effects of parents on children at the age threshold

	+/- 2 years window around the age threshold	**+/- 3 years window around the age threshold**
Treatment spillover effect (λ^c)	-0.040*	-0.037**
Standard error	(0.022)	(0.016)
N	3112	4720

Notes: See text and Section 3.2 for details on the RD set-up. Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. We use linear trends in program age that can differ at each side of the cutoff. Control variables include dummies for gender, member of risk group based on existing disorders, population density, chronic illness, education level, number of household members, family type, influenza season (see also Table 3.1). There are separate dummies for child gender and the oldest parent's gender, and there is a child age dummy for every age. OLS regression estimates are reported that use sample weights and cluster standard errors at the wave-municipality level to mimic the sample design. Significance level: * p<0.10, ** p<0.05, *** p<0.01.

The reduced form estimates of the spillover effects in equation (3.4) are shown in Table 3.3. We conclude that the RD estimate of a child's decision to vaccinate against influenza at the 65-age threshold of the oldest parent's program age is -4 percentage points in a 2 year window and -3.7 percentage points in a 3 year window.[45] The estimate is significant at a 10% significance level using the smaller window and precision increases as more observations are used. The regression-based results match the visual discontinuity in Figure 3.4. Treatment of the parents at program age 65 leads to a decrease in influenza vaccination among their adult children.

3.4.3 Further effects

While the free vaccination policy has a large beneficial impact on the immunization behavior at age 65, and substantial negative spillover effects among the adult children, these effects are silent about the effects on sickness absence, morbidity, medical care consumption and mortality. Arguably, policy makers are more concerned with these effects, rather than with the simple and direct effect on vaccination behavior. We study these effects in this section. The interpretation of the effects however differs from the effects presented in section 3.4.2. Both the estimates in this section and in section 3.4.2 are ITT estimates, but the estimates in this section – as explained in section 3.2.5 – are the result of policy induced changes in the vaccination behavior of both the parents *and* their adult children; and it is not possible to disentangle their separate contributions.

[45] Marginal effects obtained from probit regressions show similar effects: -0.031 and -0.030 for respectively the smaller and larger window.

3.4.3.1 Policy effects at age 65

In this section, we consider the impact estimates of the vaccination policy on morbidity, medicine and medical care use, and mortality at the 65-age threshold. If influenza vaccination affects these outcomes, then it seems only reasonable that the effect size should be larger during those months that influenza prevalence is high. We expect little or no effect in other months since influenza is an acute infectious disease and potential effects on morbidity/care/mortality occur in the short run. The estimates in Table 3.4 and the Appendix Figures 3.12 to 3.28 reflect the policy impact during the potential influenza-epidemic months of November to May for morbidity and health care usage. Since individuals do not immediately die from influenza(-related complications), but usually within a month, we consider the period December to June for the mortality indicators. In case of the mortality indicators, the estimates exactly correspond to mortality during the months December-June, but the other dependent variables have recall periods of 1 to 2 months. We use January to June for influenza-like symptoms and GP/specialist visits which have a 2 month recall period; and December to June for medicine use which has a 1 month recall. While subdivision into epidemic and non-epidemic periods makes a lot of sense, it runs the danger of losing precision in the estimates due to insufficiently large sample sizes (except for mortality where we use the administrative data which covers the entire Dutch population). Table 3.4 shows that, despite this potential worry, several effects are estimated with high precision.[46]

The estimates in column 2 of Table 3.4 show that those receiving an invitation for a free influenza vaccination show an almost 14 percentage points reduction in the use of prescribed medicines and GP visits, and a reduction of 0.8/100,000 deaths due to pneumonia and influenza and 1.1 out of 100,000 due to respiratory diseases. The estimates also reveal 1.7 out of 100,000 averted deaths due to respiratory and circulatory diseases, but this effect is estimated with less precision.[47] At the same time, we observe no significant (and much smaller) effects on influenza-like symptoms, non-prescribed medication, visits

[46] We have also estimated 132 separate models for the 4 mortality indicators for each month-year combination, i.e. 11 influenza seasons times 12 months. This has the advantage that it allows comparing the estimated month-year-specific effect sizes with month-year-specific influenza incidence rates obtained from influenza surveillance data (Donker, 2010). Moreover, as shown by Ward (2014), influenza vaccination will only adequately work when the match between the prevailing influenza viruses and the virus strains used in the influenza vaccine is good, which is only revealed ex-post. Hence, one expects large effect sizes of influenza vaccination in months with a high influenza prevalence and a good match of the vaccine, and smaller or no effects in the other months. Our results, available on request, show very noisy patterns over the months-years. This suggests that, despite using administrative data which covers the entire Dutch population, there is insufficient variation across months/years at the age threshold.

[47] The RD design deals with issues of selective mortality (or survivor bias) by nature.

to medical specialists, and all-cause mortality. Estimates obtained from a +/- 3 year window show smaller and slightly less precisely estimated effect sizes, but the same general picture emerges (see Appendix Table 3.8). Estimates obtained from identical models on the non-epidemic period reveal much smaller and mostly insignificant effect sizes.[48,49] The story that seems to emerge from these results suggests that the impact of the free vaccination policy on influenza-like symptoms is limited at age 65, but that the more serious consequences of influenza infection, i.e. GP visits and mortality, are averted. The contrast between the results for prescribed and non-prescribed, and GP and medical specialist visits are in line with this interpretation. For medicines, the interpretation of the different effects is relatively straightforward as individuals can only consume prescribed medicines after having visited a GP.[50] The insignificant and much smaller effect size of visits to the medical specialist (as compared to GP visits) is in line with the acute nature of an influenza infection.

3.4.3.2 Spillover effects among adult children

The third column in Table 3.4 shows the impact estimates on the same set of outcomes and influenza-related sickness absence[51] during the influenza-epidemic months for the children with parents at the 65-age threshold. We find relatively large effect sizes for influenza-like symptoms (7 to 10 percentage points) and sickness absence (7.5 percentage points), but for sickness absence the effect turns only significant in the +/- 3 year window (see Appendix Table 3.8). All other estimated effect sizes are insignificant. When compar-

[48] For influenza-like symptoms, medication use and medical care use, all estimated effects are (positive and) non significant for the 3 year window. A similar pattern emerges for the 2 year window, but here GP visits and visits to the medical specialist are significant at respectively the 5 and 10% significance level. More importantly, we find that the difference between the coefficients in the epidemic and non-epidemic periods is statistically significant for GP visits and prescribed medicines at the 5% level in both windows and amounts to round respectively 40 and 30 percentage points. The other coefficient differences are not statistically significant. For the mortality indicators, we did not estimate separate models for the non-epidemic period, but rather estimated models on the full influenza season (i.e. from September to August). We do this because it is unclear how to classify individuals that survive the first months of the non-epidemic period (September-November), but who die in the last months of the non-epidemic period (July-August). This problem does not occur for the indicators derived from HIS, because we only observe individuals who are alive in HIS. Estimates derived from the full September-August influenza season only show (positive) significant effects for all-cause mortality confirming that influenza vaccination protects against influenza related deaths during the epidemic period only.

[49] We also find, not unexpectedly, that all 4 mortality indicators decline over time. In addition, we find higher mortality rates during the influenza seasons of 1999/2000 and 2004/2005, which had much higher than average levels of influenza prevalence.

[50] It could also be that the insignificance of the non-prescribed medicines reflects that medicines for influenza-like symptoms only constitute a minor share of all consumed non-prescribed medicines, but this cannot be checked with our data.

[51] We consider the survey months January to June for sickness absence, since the question refers to the last two months.

Table 3.4: RD estimates for other outcomes on parents and children during the influenza epidemic period (in a +/- 2 year window)

	Parents	**Children**
Influenza-like symptoms	0.004	0.109
	(0.071)	(0.070)
Non-prescribed medication	-0.071	-0.048
	(0.050)	(0.045)
Prescribed medication	-0.139	-0.009
	(0.044)***	(0.040)
GP visits	-0.137	-0.029
	(0.056)**	(0.041)
Visits to medical specialist	-0.014	0.038
	(0.047)	(0.046)
Sickness absence		0.071
		(0.048)
Mortality: pneumonia and influenza	-0.000082	0.000005
	(0.000038)**	(0.000006)
Mortality: respiratory diseases	-0.000113	0.000008
	(0.000059)*	(0.000009)
Mortality: respiratory and circulatory diseases	-0.000171	0.000006
	(0.000102)*	(0.000017)
Mortality: all-cause mortality	-0.000008	-0.000010
	(0.000148)	(0.000034)

Notes: See text and Section 3.2 for details on the RD set-up. We use linear trends in program age that can differ at each side of the cutoff. Control variables include dummies for gender, member of risk group based on existing disorders, population density, chronic illness, education level, number of household members, family type and influenza season for outcomes 1 to 6. Control variables for mortality include gender and influenza season. In addition all regressions for children include dummies for children's program age per year and parent's gender. Standard errors between brackets. Regression estimates with HIS use sample weights and cluster standard errors at the wave-municipality level to mimic the sample design. Regressions on administrative data apply robust standard errors. The influenza epidemic period is defined as November-May: (a) influenza-like symptoms, sickness absence and GP/specialist visits refer to the last two months, so the survey months January to June are considered; (b) medicine use refers to the last month, so the survey months December to June are used; and (c) mortality refers to the actual months December-June since most influenza-related deaths occur within one month of the original infection. Significance level: * $p<0.10$, ** $p<0.05$, *** $p<0.01$.

ing the estimates for the epidemic months with those of the non-epidemic months, we find that influenza-like symptoms and sickness absence are statistically significantly (at the 5% level) higher in the epidemic periods.[52] All other coefficient differences between epidemic and non-epidemic periods are not significant. These findings suggest that the free vaccination program at age 65 has important spillover effects beyond the vaccination behavior of the adult children: there seems to be little or no impact on medicine use, medical care visits and influenza-related mortality, but there is a very substantial increase of influenza-like symptoms and sickness absence.[53] The finding that there is no effect on GP visits, despite the increase in symptoms and sickness absence, is in line with the low mean number of GP visits in the Netherlands compared to other OECD countries (Van Doorslaer et al., 2006).[54]

3.4.4 Robustness analysis: Internal validity of our findings

The estimates presented in sections 3.4.1-3.4.3 represent 'intentions to treat' (ITT). These ITT's are internally valid when the assumption of independence, which means that treatment is random, is satisfied. In our RD setting, this implies that the only discontinuous change within a small window around the 65-age threshold is the change in vaccination policy. As mentioned in section 3.2.3, there is little a priori reason to believe this assumption does not hold: age cannot be manipulated; anticipation makes little sense as influenza vaccination needs to be taken yearly to be protective; and potential interference with age-triggered eligibility for other programs (such as pension benefits) should be limited, since eligibility for free influenza vaccination is determined by being 65 or older on May 1st.

We have done several tests that confirm the independence assumption. First, there should be no discontinuous differences between the characteristics of individuals just below and above the age threshold. We ran RD models with the child and parental control covariates) as the dependent variables. Appendix Tables 3.9 and 3.10 show that none of the RD estimates of the child covariates is significantly different from zero. A similar picture emerges for the parents sample, with the exception of family type (couple versus single) and population density of the place of residence which turn significant at the 5%

[52] The coefficients are respecively 15 and 19 percentage points higher in the epidemic versus non-epidemic periods.

[53] We also estimated month-year specific models, but the resulting estimates, which are available on request, reveal even noisier patterns compared to those for the parents at the 65-age threshold;

[54] In addition, employers do not require a GP to confirm sick leave, a practice that is compulsory in several European countries.

and 10% level, respectively. The corresponding RD graphs, which are available on request, nevertheless suggest that the unbalancedness of family type and population density around the 65-age threshold vanish for larger windows. In addition, controlling (or not) for family type and population density do not strongly affect the estimated treatment effects in Tables 3.2-3.4 (see also discussion and Table 3.5 below). We conclude that covariates in the parents sample are reasonably balanced around the 65-age threshold. We did not check balancedness of covariates in the parents and child administrative samples as the covariates are limited to gender (and child age in the child sample) which we do not expect to be unbalanced around the age threshold.

A second test checks whether the density of age is balanced at both sides of the age threshold. Again there is no a priori reason to expect a violation in the parents HIS sample and the administrative datasets, but it could potentially play a role in the child HIS dataset when parental age at time of the child's birth is systematically related to a child's later influenza decision. A frequency histogram of the density of parental age in the child sample of HIS is relatively smooth, but shows a slightly lower frequency at the age of 66 (see Appendix Figure 3.11). If this deviation is random, then the estimates presented in sections 3.4.2-3.4.3.2 are valid, but unfortunately this is untestable. Instead, we can calculate bounds on the RD estimates in Figures 3.3-3.4 and Tables 3.2-3.4 assuming that all 'missing' individuals with parents of 66 are compliers or non-compliers. In practice, we weigh the compliers or the non-compliers in the RD estimation procedure such that the frequency of individuals with parents of 66 becomes identical to the average frequency of the other age groups in Appendix Figure 3.11. Despite the fact that some of these hypothetical states are very unrealistic[55], the derived bounds are very small and zero is never included in these bounds, essentially meaning that our estimates pass this test.

We have also tested the sensitivity of the estimates to the assumptions imposed to capture the trends in the outcome variables, i.e. the choice of parametric form and the window size. In Appendix Table 3.11, the window size around the age threshold is varied for the models of vaccination take-up with linear trends in program age. We conclude that the estimates are relatively stable (in particular the ones for the spillover effects onto the children) for different window specifications, but the direct policy effects at age 65 are larger for wider windows, and the opposite trend is observed for the negative spillover effects among the adult children. Table 3.5 (row "quadratic trends") reveals the impact

[55] In particular, when the compliers or the non-compliers constitute a very small group of the individuals in the sample with parents aged 66.

Table 3.5: Robustness checks in a +/- 3 year window around 65-age-threshold

	Parents	**Children**
Baseline effect	0.184***	-0.037**
	(0.026)	(0.016)
No controls	0.179***	-0.046***
	(0.026)	(0.016)
Region-time fixed effects	0.185***	-0.036**
	(0.026)	(0.016)
Clustering at program age	0.184***	-0.037**
	(0.027)	(0.013)
Quadratic trend	0.158**	-0.042
	(0.062)	(0.040)

Notes: See text and Section 3.2 for details on the RD set-up. Unless otherwise specified, we use linear trends in program age that can differ at each side of the cutoff. Control variables include dummies for gender, member of risk group based on existing disorders, population density,chronic illness, education level, number of household members, family type, influenza season. In addition the analysis for adult children includes dummies for children's program age per year and parent's gender. OLS regression estimates are reported that use sample weights and cluster standard errors at the wave-municipality level to mimic the sample design. Standard errors between parentheses. Significance level: * p<0.10, ** p<0.05, *** p<0.01.

of experimenting with quadratic trends instead of the linear trends (see also Figures 3.5-3.10). Note that the small window and the yearly bins do not provide us with many data points to estimate polynomial trends. Table 3.5 shows that the estimates are not very much affected by the quadratic trend, but they are less precisely measured. The spillover effects are no longer significant at the 10% level.

In addition, Table 3.5 reports alternative specifications keeping the +/- 3 year window fixed. The first row reproduces the baseline results. A first alternative is to exclude the control variables from the regression. We however keep the fixed effects for the influenza season and children's age in the regression specification, since these variables capture trends in our pooled cross-section data. Second, we include province fixed effects per influenza season. There are 12 provinces in the Netherlands with varying population sizes (ranging between 400,000 to 3,500,000 individuals). The region-time fixed effects are a flexible way to capture variation in our data that may originate from e.g. regional GP medical practices, a different intensity and spread of previous influenza seasons across provinces, regional variation in incidence of other infectious diseases, regional information campaigns etc. Our results remain unaffected. Next, we apply a different clustering of the standard errors. Up to now, we have allowed the standard errors to be clustered at the municipality-wave level to mimic the sampling procedure. Alternatively, we can cluster at the level of the running variable, i.e. the program age of the parents. We observe that the

standard errors are somewhat larger using the alternative clustering, but the estimates remain significant at the 5% level.

Our general conclusion is that the list of tests support the findings reported in sections 3.4.1-3.4.3. These tests suffice for the internal validity of our results since we only report ITT estimates, but are uninformative on the channels underlying the treatment effects of crossing the 65-age threshold. All one can conclude from these tests is that crossing the 65-age threshold has an internally valid causal impact on the dependent variables, but not that these effects *only* reflect the impact of the free influenza vaccination policy. A potential worry is that our estimates are partly driven by age-triggered eligibility for other programs, such as pensions for which Dutchmen turn eligible at age 65 in the Netherlands. As explained before, there is little reason to expect an effect since the age eligibility of the free influenza vaccination policy does not coincide with the day one turns 65. Moreover, monetary aspects of being retired might not matter for vaccination take-up since influenza vaccination is free; and changes in time costs might be limited since GPs organize flexible hours to receive the influenza vaccination. Nevertheless, we did run RD models with being retired as the dependent variable. We defined being retired in a broad sense, i.e. individuals not working[56], to make sure that we consider the monetary and time aspects of being retired/not working. The resulting RD estimate shows a significant increase at the 65-age threshold, but the effect is very small and hence unlikely to be driving the jump in vaccination take-up at age 65. We also ran RD models of vaccination take-up at age 65 for the subgroup of retired individuals only. The resulting estimates are very similar to, and not statistically different from, the estimates in section 3.4.1. In addition, we ran two types of placebo RD regressions. The first replaces the dependent variable by indicators of preventive care use to exclude the possibility that crossing the 65-age threshold makes one more or less likely to use preventive care. The resulting estimates confirm that our RD design is not picking up patterns of preventive care use.[57] The second type of placebo RD regressions sticks to vaccination take-up as the dependent variable, but changes the age threshold to 62 (to make sure that the true cutoff of 65 is not in the window). None of the resulting estimates is significant.

[56] The non-working category consists of three broad categories: (1) individuals that are 65 or more; (2) individuals that are unemployed, full-time students, or not working; and (3) individuals that receive disability insurance benefits or pension benefits.

[57] We ran models for the direct policy effects and the spillover effects with a +/- 2 and 3 year window. We used the following dependent indicators: (a) having diabetes; (b) exercising at least once a month; (c) BMI (to capture the combination of physical exercise and food intake); (d) engaging in breast cancer screening. None of the resulting RD estimates was statistically significant, except for BMI in the child sample in the +/- 2 year window which turned significant at the 10% level (not significant in the +/- 3 year window).

3.5 Results: spillover effect channels

In this section, we dig deeper into the spillover effects of the free influenza vaccination policy. Section 3.4.2 showed negative spillover effects reducing the vaccination coverage rate among the adult children from around 9% to 5%, and section 3.4.3.2 confirmed increased prevalence of influenza-like symptoms and sickness absence. These effects are substantial, but it is less clear what channels are causally linking the free influenza vaccination policy to reduced vaccination coverage among the adult children. The question of unraveling the causal link with influenza-like symptoms and sickness absence is further complicated – as explained in section 3.2.5 – by the fact that both parental and child vaccination take-up might play a role. Since it is impossible to unravel their separate impact – without additional exogenous variation that drives child vaccination take-up –, we limit this section to the channels leading to reduced vaccination coverage among the children.

This does not mean that the channels underlying vaccination take up among the children are easy to uncover. The sensitivity analyses in section 3.4.4 suggest that vaccination take up among the children is driven by increased vaccination take-up among the parents at age 65. We believe this is a reasonable explanation (also because the vaccination cost of the children remains unaffected by the free vaccination coverage), but our data does not allow excluding alternative explanations for the negative spillovers.[58] For example, children whose parents where already vaccinating before crossing the age threshold might have reduced their vaccination take-up. Similarly, children might become more susceptible to campaigns on influenza vaccination as soon as their parents have reached the age of 65. These two alternative explanations, in combination with our data limitations, show that we can at best provide suggestive evidence on the underlying mechanisms.

The negative spillover effect in section 3.4.2 should not be uniform across all individuals, and the estimates in Table 3.3 might conceal heterogeneity in the treatment effect. Heterogeneity (or the lack thereof) can provide suggestive evidence about the mechanisms/channels at work. Since we do not have direct information on vaccination take-up of parents in our child sample, we obtain heterogenous treatment effects by estimating pooled regression models with interactions between children's characteristics on the one hand, and the treatment effect and the trends at both sides of the age threshold on the other hand.

A potential explanation why children forgo immunization when their parents vaccinate could result from epidemiological externalities within the family. As explained before, this

[58] This derives from the fact that vaccination behavior of the parents is not included in the child sample.

Table 3.6: Heterogeneity in negative spillover effect (+/- 3 year window around 65-age threshold)

	Children
Baseline	-0.037 (0.016)**
Panel A: Education level	
Education level 1: primary	-0.183 (0.108)*
Education level 2: lower secondary	-0.025 (0.037)
Education level 3: upper secondary	-0.039 (0.023)*
Education level 4: post-secondary	-0.019 (0.025)
Panel B: Distance with parents' municipality of residence	
Same municipality	-0.049 (0.016)***
Distance 0 to 20 km	-0.041 (0.020)**
Distance 20 to 60 km	-0.024 (0.024)
Distance 60 km or more	-0.006 (0.030)
Panel C: Member of risk group based on existing disorder	
Member of risk group	-0.129 (0.105)
Not a member of risk group	-0.029 (0.014)**
Panel D: Employed as health care worker	
Employed as health care worker	-0.051 (0.023)**
Not employed as health care worker	-0.036 (0.016)**
Panel E: Family type	
Single person	-0.040 (0.041)
Couple	-0.057 (0.035)*
Household with children	-0.030 (0.020)
Panel F: Employment	
Not employed	-0.030 (0.031)
Salaried employment	-0.039 (0.016)**
Self-employed	-0.037 (0.026)

Notes: See text and Section 3.2 for details on the RD set-up. We use linear trends in program age that can differ at each side of the cutoff. Control variables include dummies for gender, member of risk group based on existing disorders, population density, chronic illness, education level, number of household members, family type, influenza season, dummies for children's program age per year and parent's gender. OLS regression estimates are reported that use sample weights and cluster standard errors at the wave-municipality level to mimic the sample design. Standard errors between parentheses. Significance level: * $p<0.10$, ** $p<0.05$, *** $p<0.01$.

is different from the externalities considered by Ward (2014). If this were the case, we would expect the externalities to be relatively more important for individuals who (i) have regular physical contact with their parents, (ii) are less likely to be infected from other sources, and (iii) are less inclined to take up influenza for other (important) reasons, such as their own health. While we cannot explicitly test these hypotheses, we can provide suggestive evidence using proxy variables. Point (i) is addressed in panel B of Table 3.6. We test the idea that individuals who live close to their parents – and therefore potentially have more physical contact – might have a more negative spillover effect. Point (ii) is dealt with in panels D, E and F. Panel E considers the idea that adult children who have kids themselves are probably less worried to be infected through contacts with their parents than through their children, in particular since young children are more important influenza transmitters than the elderly (Galvani et al., 2007; Halloran & Longini, 2006). Panel D and F test the same idea for respectively health care workers who tend to have regular contact with sick individuals and the work environment which is a likely disease intermediary. With regard to point (iii), we test in panel C the idea that members in the high-risk group based on existing disorders are less affected by epidemiological externalities, since they have a stronger personal incentive to be vaccinated. Overall, our findings in Table 3.6 are not very much in favor of the externality explanation. While we cannot reject that externalities within the family matter, our results suggest that they play a minor role at most. Only two of the 5 panels show the expected patterns (panel B and E), and none of the differences in spillover effects within panels are statistically significant (not even at the 20% significance level).

Alternatively, learning about costs related to vaccination could explain our results. What costs should we think of? The costs are revealed to individuals when their oldest parent crosses the age threshold, so upon receiving the invitation or related to the vaccination (procedure) itself. It appears that the costs are less relevant for the parents themselves, since vaccination participation jumps upwards at the cutoff and further increases afterwards.

We conclude from previous studies in the Netherlands that analyze the motivation for non-vaccination against seasonal or pandemic influenza, that not being part of the target group is an important reason not to vaccinate. Using a sample of 4000 Dutch citizens, Krooneman & Verheij (2003) show that it is the principal reason not to vaccinate for adults without chronic disease (indicated by 56% of the individuals) and the second most important reason for individuals that belong to the high-risk group (reported by 23% of the individuals at risk). The latter is surprising, and indicates that an important share

Table 3.7: Heterogeneity in treatment effect (+/- 3 year window around 65-age threshold)

	Parents	**Children**
Month of birth between November and April	0.141 (0.040)***	-0.054 (0.023)**
Month of birth between May and October	0.198 (0.040)***	-0.027 (0.024)

Notes: See text and Section 3.2 for details on the RD set-up. We use linear trends in program age that can differ at each side of the cutoff. Control variables include dummies for gender, member of risk group based on existing disorders, population density, chronic illness, education level, number of household members, family type, influenza season for the first stage and the reduced form. In addition the reduced form includes dummies for children's program age per year and parent's gender. OLS regression estimates are reported that use sample weights and cluster standard errors at the wave-municipality level to mimic the sample design. Standard errors between parentheses. Significance level: * p<0.10, ** p<0.05, *** p<0.01.

of the individuals at risk might not be fully aware that they are being targeted by the vaccination program or prefer not to participate. Bults et al. (2010) and van der Weerd et al. (2011) analyze reasons not to vaccinate against the Mexican flu. Van der Weerd et al. (2011) analyze a nationally representative sample of 8060 individuals contacted in three phases: the early phase, the pandemic alert phase and the official epidemic. They show that information campaigns reached the general population, with 58% of the individuals informed in phase 2 and 85% in phase 3.[59] Among the reasons to refuse vaccination, not being part of the high-risk group was the 6^{th} most important reason in phase 2, but as information coverage increased, it became the second most important reason in phase 3. Bults et al. (2010) also underline the importance of being targeted for vaccination take-up.

Next, we look at the invitation letters drawn up by the Dutch college of general practitioners that serve as example for the GPs to personally invite the population at risk.[60] The last three example invitation letters (of 2009, 2011 and 2012) are obtained.[61] The letters underline that vaccination offers protection against influenza, that vaccination is free of charge and provide the following definition of the target group: "Individuals aged 60 or more[62], and individuals of all ages with heart, lung or renal disease, diabetes or with immune dysfunctions, are at increased risk to fall seriously ill due to influenza. You

[59] Note that Dutch citizens are among the European citizens that put most trust in their national and regional governments (see Eurobarometer) and attach importance to governmental information on health issues (van der Weerd et al. (2011), Bults et al. (2010)).

[60] Note that the GP is not obligated to follow the example invitation letter, and is free to formulate an alternative invitation letter or not to send out an invitation at all. As we discussed in footnote 17, most GPs do send out a personal invitation.

[61] We contacted the Dutch college of general practitioners to obtain all example invitation letters between 1996 and 2012, but only the invitation letters of 2009, 2011 and 2012 were recovered.

[62] Since 2008, the lower age threshold of 60 was in place.

belong to one of these groups. That is why you are eligible for influenza vaccination." In 2011 and 2012, the invitation letter mentions for a second time: "Influenza vaccination is for individuals at increased risk to fall seriously ill due to influenza." We conclude that, first, it is made very explicit who is targeted and who is not. Second, the reference to the age threshold in the letter can be confusing for individuals who receive the letter for the first time. The invitations are received around September/October. Since program age is determined as of May 1st in the following year, the real age of individuals whose month of birth is between November and April falls below the age threshold. For this group, the receipt of the letter can come across as a mistake made in the invitation procedure.

The explicit framing of the target group in the invitation letter can be picked up by the children of eligible individuals as a reason not to vaccinate and might induce a social stigma cost to vaccinate. Social stigma in this context can be more important for lower educated than for higher educated individuals (Panel A in Table 3.6). The latter might be less impressed by the wording of the invitation letter or generally better informed about the program and the possibilities to vaccinate when not targeted. More frequent regular contact can facilitate the exchange of information and explain why we find a stronger effect if parents and children live close to one another (panel B in Table 3.6). Finally, in order to analyze whether or not the information in the invitation letter has the potential to affect individual's behavior, we analyze behavioral differences between individuals based on the oldest parent's date of birth. Remember that for parents with month of birth between November and April, the information in the letter might be confusing, which can contribute to discussing the letter more intensely with friends and family and to changing their own vaccination behavior. Table 3.7 shows that the parent's vaccination behavior is significantly lower for parents born between November and April. There is a 5 percentage points difference in take-up between both groups. In addition, the RD estimate on the adult children is almost double the size if the parent is born between November and April.

3.6 Conclusion

In this chapter, we investigated spillover effects of an extension of the target group for the Dutch influenza vaccination program on vaccination coverage of the individuals not targeted by the policy. A major reform redefined the target group in 1996. Before 1996, only individuals with specific disorders were targeted. In 1996, all healthy individuals aged 65 or more were added to the eligible population. Members of the target group qualify for free influenza vaccination and receive a personal invitation letter from their

GP.

Using a rich dataset that combines survey data on health with administrative records from Statistics Netherlands, we exploited the quasi-random variation that was introduced at age 65 by the reform. As being 65 on May 1st determines whether one receives an invitation for a free flu shot, and not the actual day one turns 65, our fuzzy RD design does not suffer from the typical concern that the discontinuity at age 65 may coincide with eligibility for other welfare programs or government transfers such as retirement benefits. Our dataset also allows to move beyond the first order effect on vaccination behavior, and in addition analyzes impacts on the arguably even more important outcomes of morbidity, medical care use, sickness absence and mortality.

Our results indicate a positive direct policy effect on vaccination coverage of the parents, accompanied by a negative spillover effect from parents to children. The vaccination policies in place increase immunization of the elderly by 18 percentage points at age 65, leading to a direct increase in vaccination rates from about 30% to 50%. Vaccination participation further increases after crossing the age threshold. The positive direct effect observed among targeted parents translates into a negative spillover effect on vaccination behavior among their children. The estimate of a child's decision to vaccinate against influenza shows a decrease in participation by 4 percentage points if the parent's age is above the threshold. In addition, we estimate that the influenza vaccination program saves 0.8 individuals out of 100,000 at the age threshold, and reduces the number of individuals consulting a GP and using prescribed medicines with 10 percentage points during the typical influenza months. Mortality and GP visits of the adult children are not affected, but the occurrence of influenza-like symptoms increases from 45% to 55% and sickness absence among this group increases with 8 percentage points (from 14% to 22%). All our results are robust to different specifications at the 65-age threshold, and robustness checks suggests that the identifying assumptions of our fuzzy RD design hold. We explore several possible channels that might generate the negative spillover effects and find suggestive evidence that a social stigma cost is revealed to adult children – who are not targeted by the vaccination program – when their oldest parent crosses the age threshold. A potential trigger for the social stigma cost is the explicit framing of the target group in the invitation letter sent out to eligible parents.

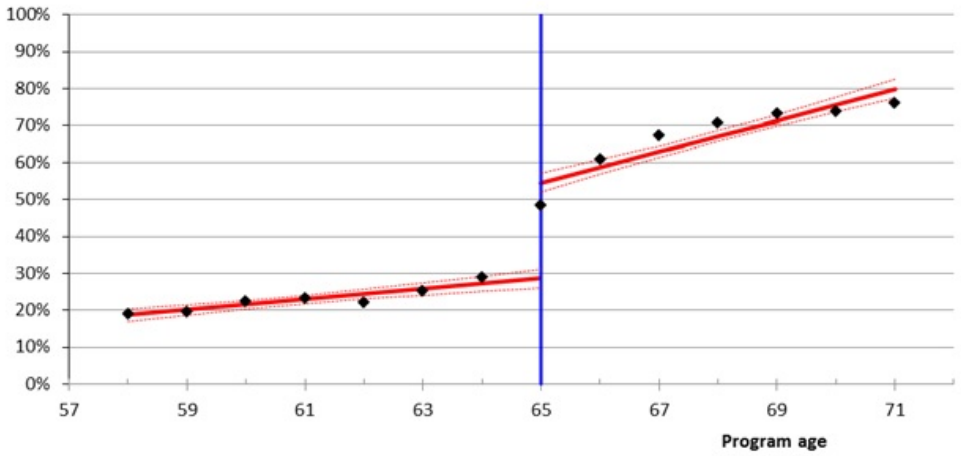
Our study improves upon existing studies in the influenza domain by considering the behavioral response in terms of vaccination take-up among the non-targeted group. It also contributes to the literature on social interactions by exploiting the quasi-random

variation created by the Dutch influenza vaccination program to overcome the typical identification challenges that arise when estimating social interaction effects. While one should be careful to extrapolate our findings outside the 65-age threshold, our findings suggest that family networks are relevant social networks and contribute to information transmission on social costs and benefits. Our results also underline the importance of public health campaigns to pay attention to the effects of information dissemination on public perceptions and attitudes on (voluntary) preventive care participation. In the case of influenza vaccination, participation outside the target group is not harmful for the personal health of the untargeted individual, it generates indirect protection to individuals at risk, it reduces productivity loss due to work absence and is considered cost-effective for large subgroups in the population. If untargeted individuals wish to finance their own vaccination, it should not be discouraged by policymakers, but directly or indirectly encouraged, e.g. by adjusting the information message that is distributed. Our findings also suggests that many (European) countries with similar influenza vaccination policies in place might face similar negative spillover effects amongst family members.

Appendix

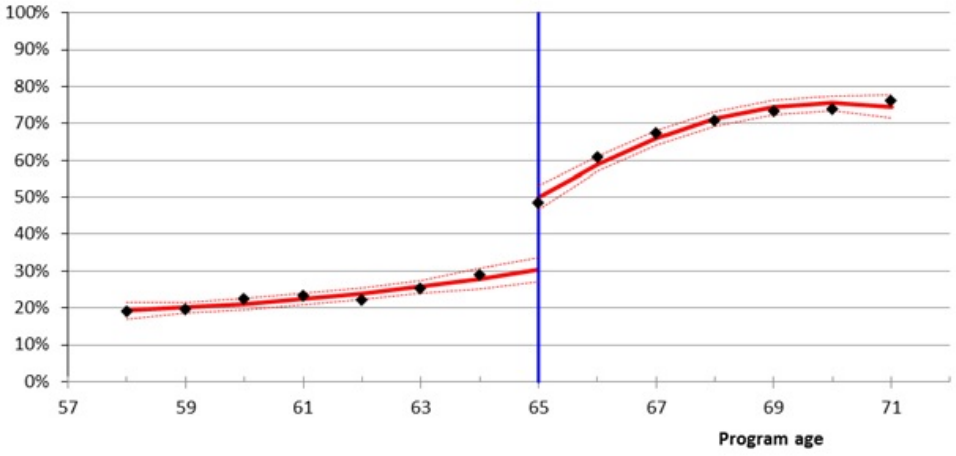
Figure 3.5: Influenza vaccination rate of individuals around the 65-age threshold (yearly bins, larger window)

Panel A: Linear trend line



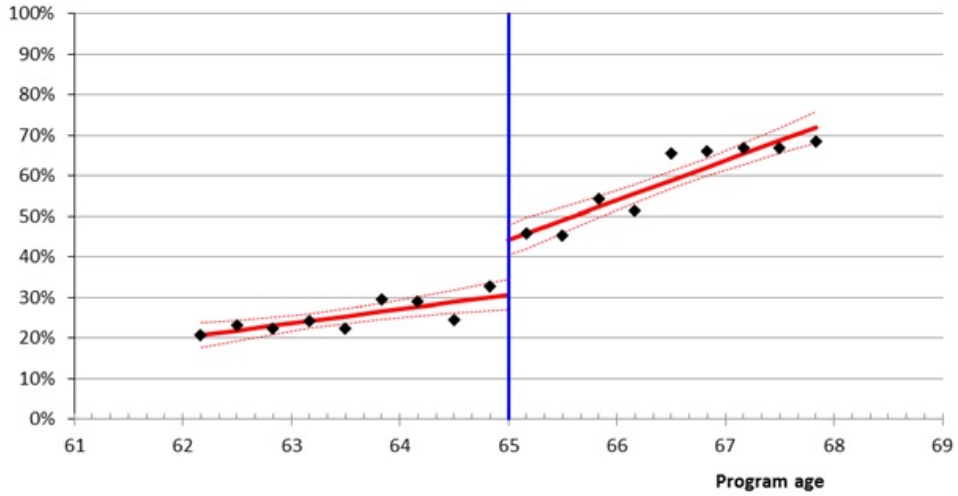
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate, grouped in yearly bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Panel B: Quadratic trend line



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate, grouped in yearly bins based on program age. The solid line shows a quadratic trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

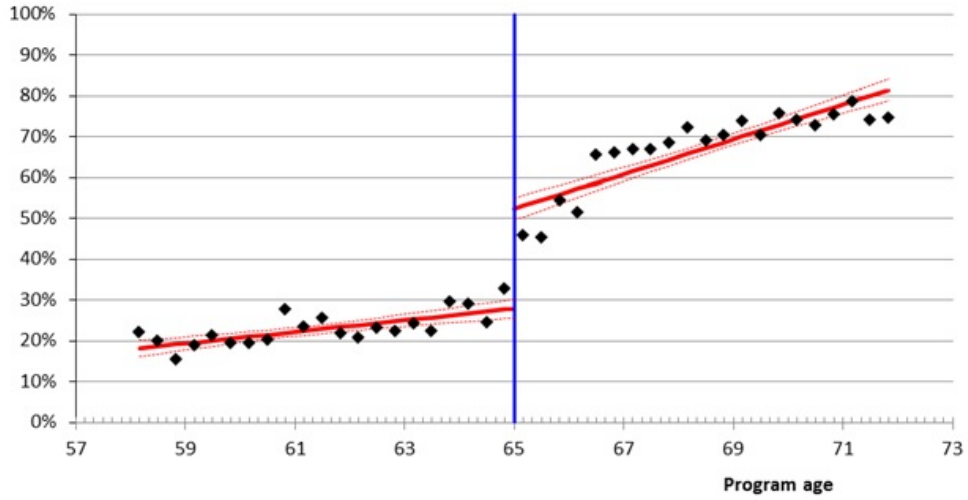
Figure 3.6: Influenza vaccination rate of individuals around the 65-age threshold (four-month bins, baseline window)



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate, grouped in four-month bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

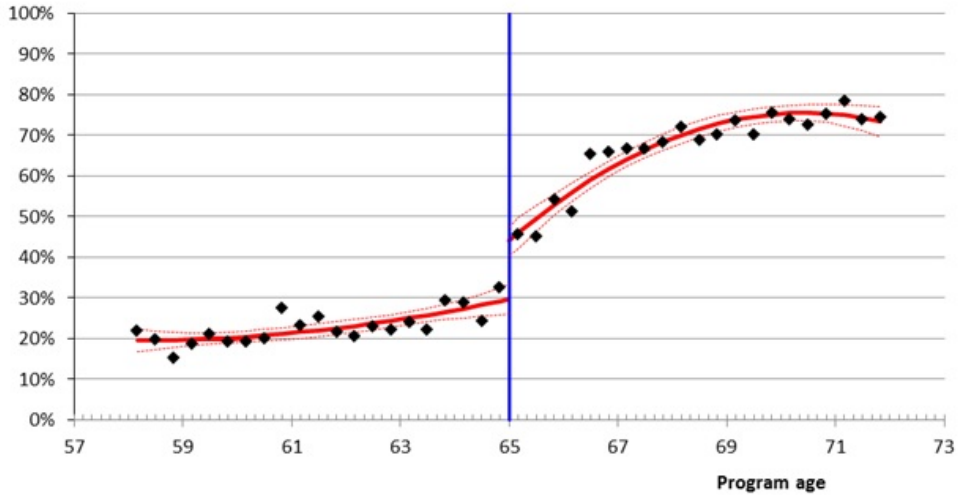
Figure 3.7: Influenza vaccination rate of individuals around the 65-age threshold (four-month bins, larger window)

Panel A: Linear trend line



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate, grouped in four-month bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

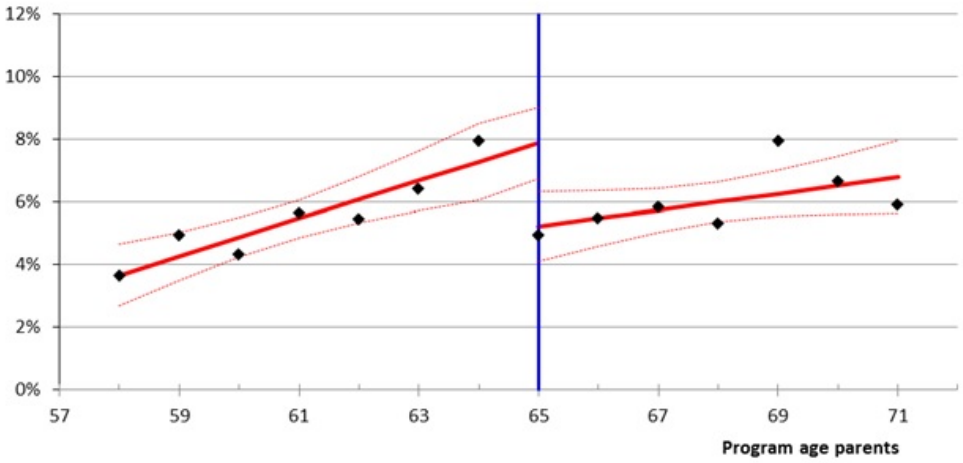
Panel B: Quadratic trend line



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate, grouped in four-month bins based on program age. The solid line shows a quadratic trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

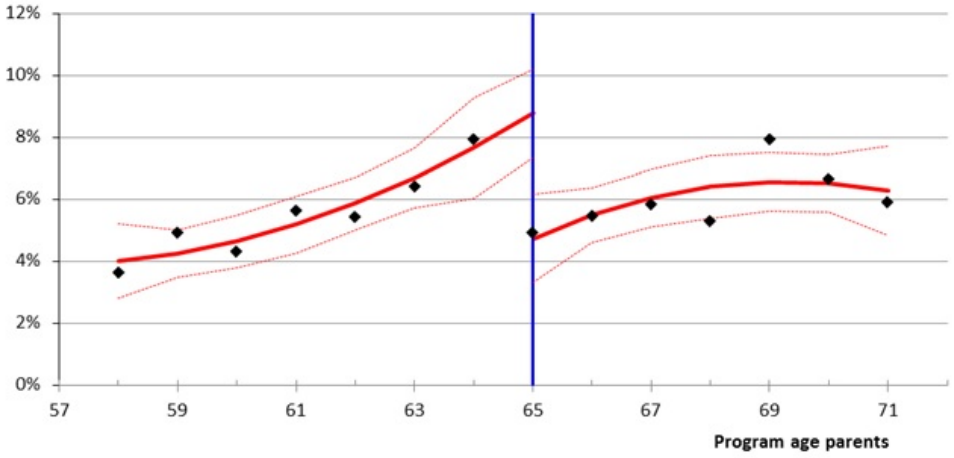
Figure 3.8: Influenza vaccination rate of children (yearly bins, larger window)

Panel A: Linear trend line



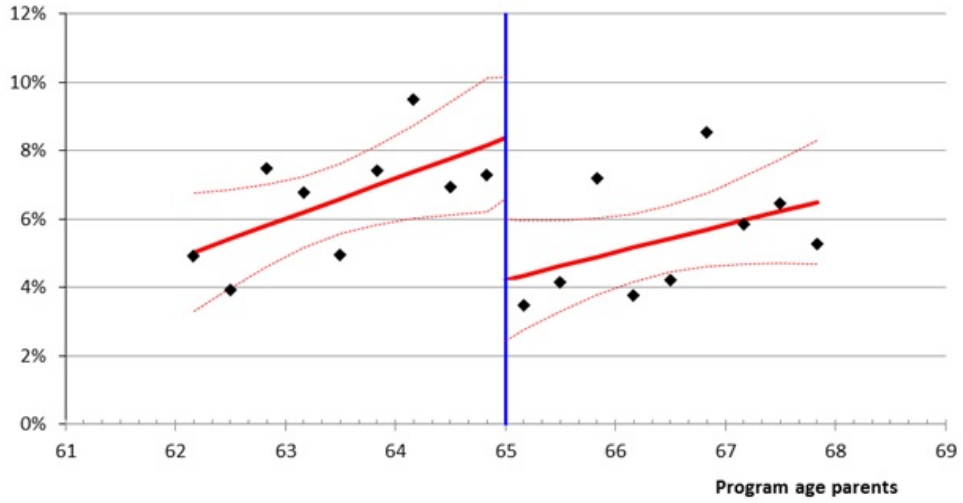
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate of children, grouped in yearly bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Panel B: Quadratic trend line



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate of adult children, grouped in yearly bins based on the oldest parent's program age. The solid line shows a quadratic trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

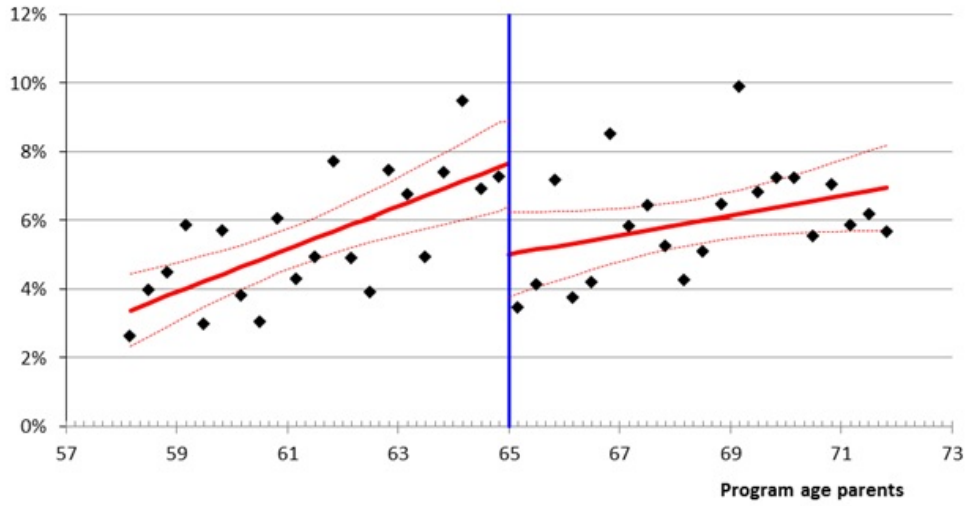
Figure 3.9: Influenza vaccination rate of children (four-month bins, baseline window)



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate of adult children, grouped in four-month bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

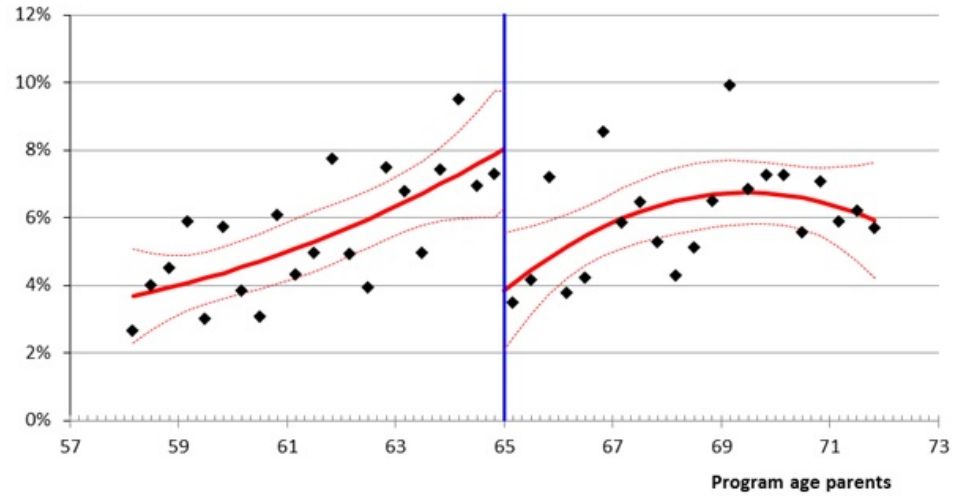
Figure 3.10: Influenza vaccination rate of children (four-month bins, larger window)

Panel A: Linear trend line



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate of children, grouped in four-month bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Panel B: Quadratic trend line



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average influenza vaccination rate of adult children, grouped in four-month bins based on the oldest parent's program age. The solid line shows a quadratic trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.11: Frequency histogram of observations of children around the parent's age threshold, influenza seasons 1997-1998 up to 2007-2008.

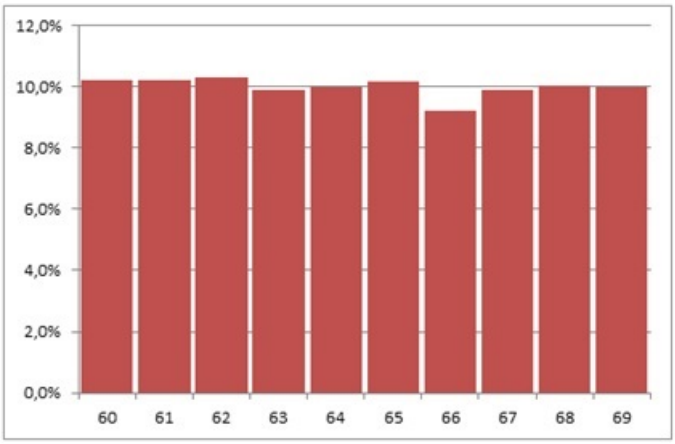
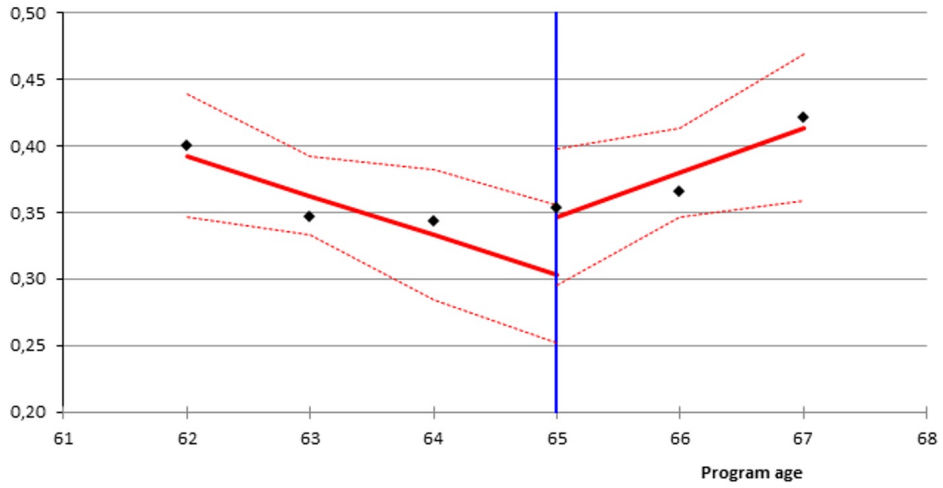
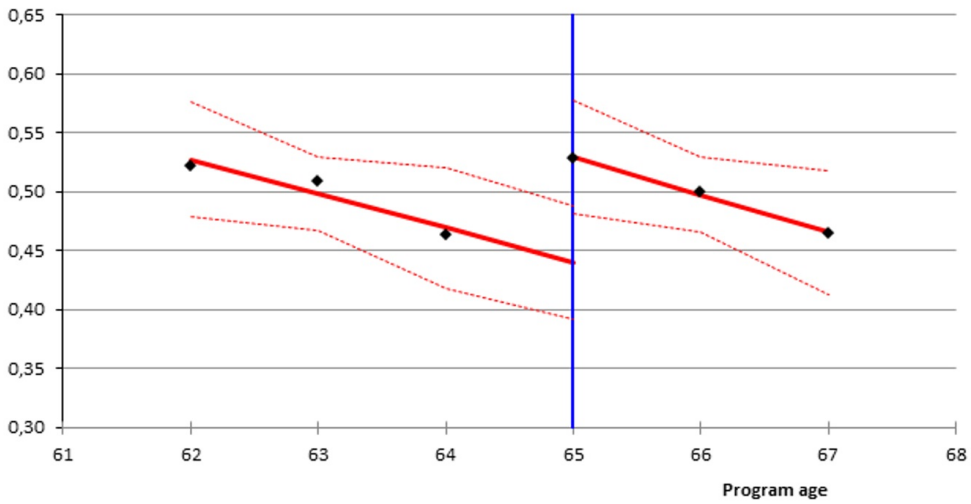


Figure 3.12: Influenza-like symptoms prevalence of individuals around the 65-age threshold



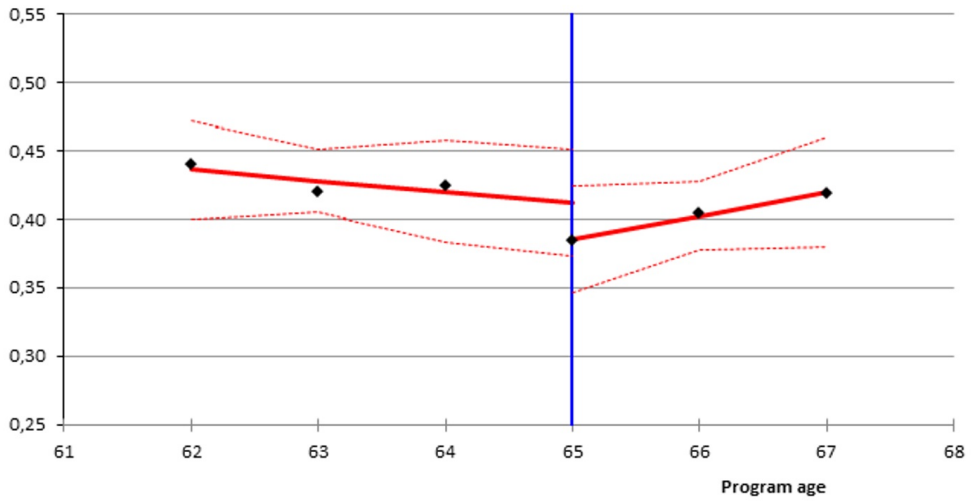
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the weighted average influenza-like illness prevalence between Nov.-May, grouped in yearly bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.13: Influenza-like symptoms prevalence of adult children with parents around the age threshold



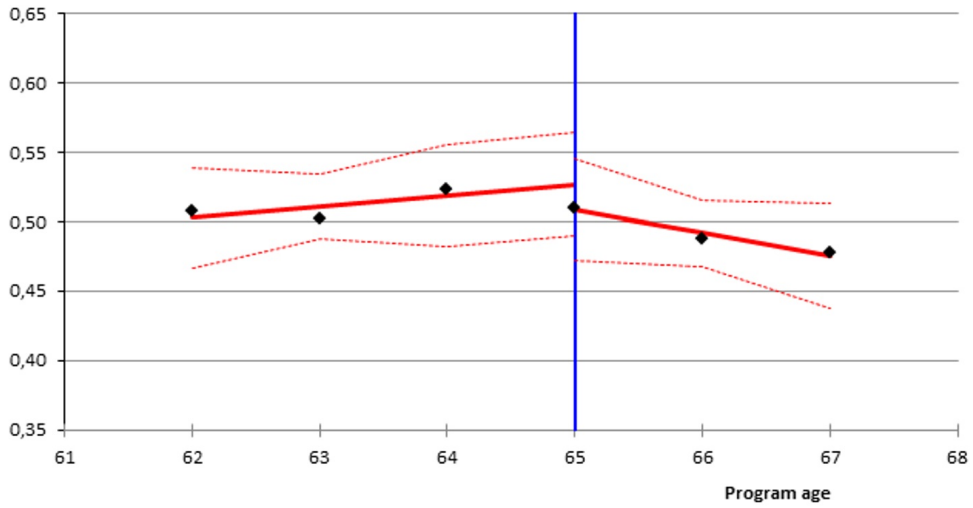
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average influenza-like illness prevalence of adult children between Nov.-May, grouped in yearly bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.14: Non-prescribed medication usage of individuals around the 65-age threshold



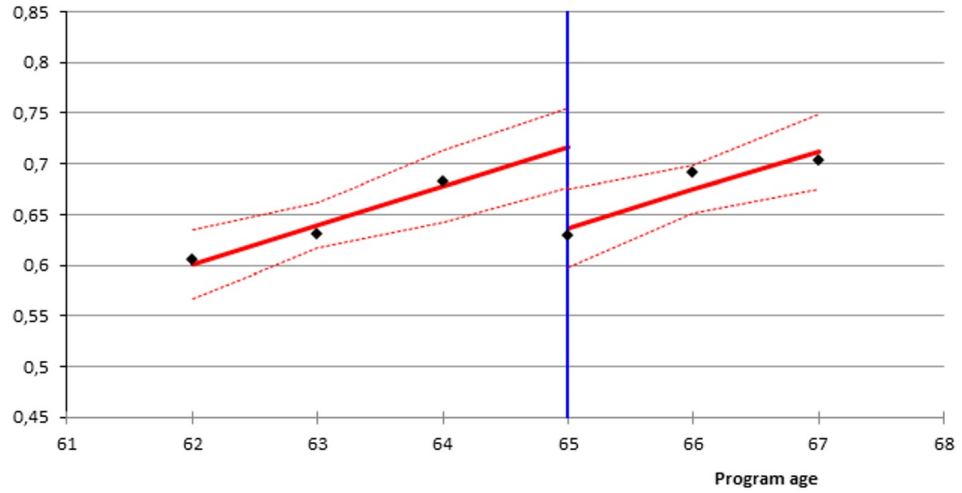
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the weighted average non-prescribed medication usage between Nov.-May, grouped in yearly bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.15: Non-prescribed medication usage of adult children with parents around the age threshold



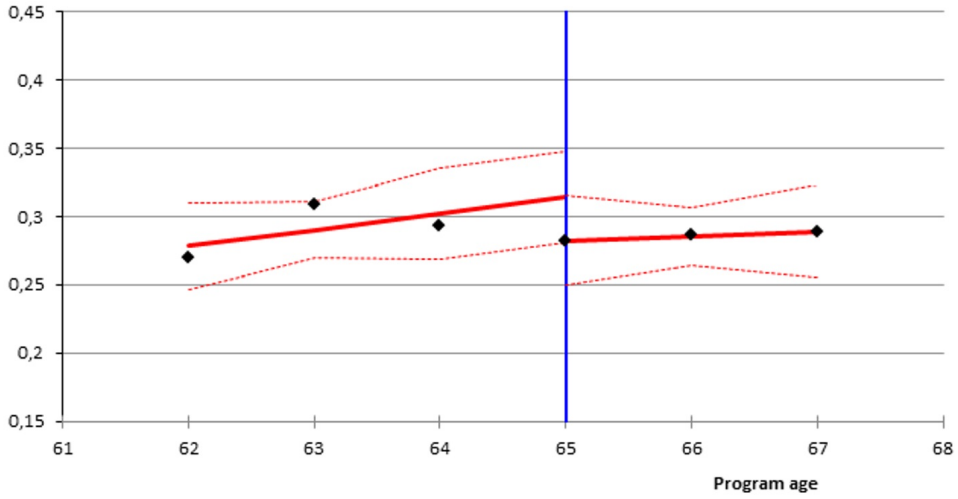
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average non-prescribed medication usage of adult children between Nov.-May, grouped in yearly bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.16: Prescribed medication usage of individuals around the 65-age threshold



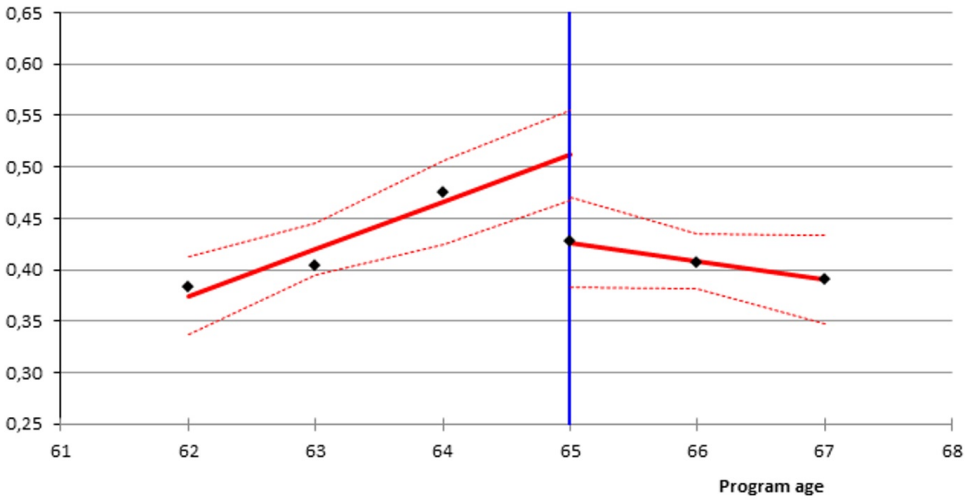
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the weighted average prescribed medication usage between Nov.-May, grouped in yearly bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.17: Prescribed medication usage of adult children with parents around the age threshold



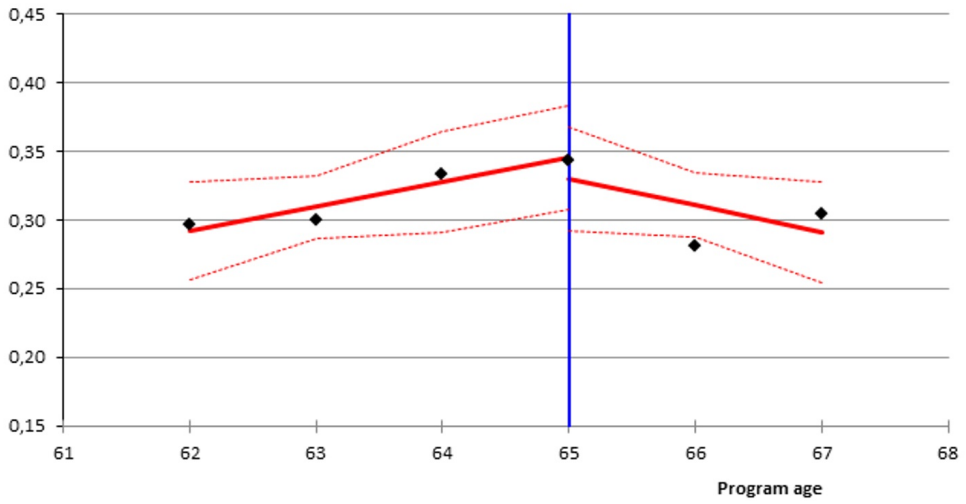
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average prescribed medication usage of adult children between Nov.-May, grouped in yearly bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.18: GP visited by individuals around the 65-age threshold



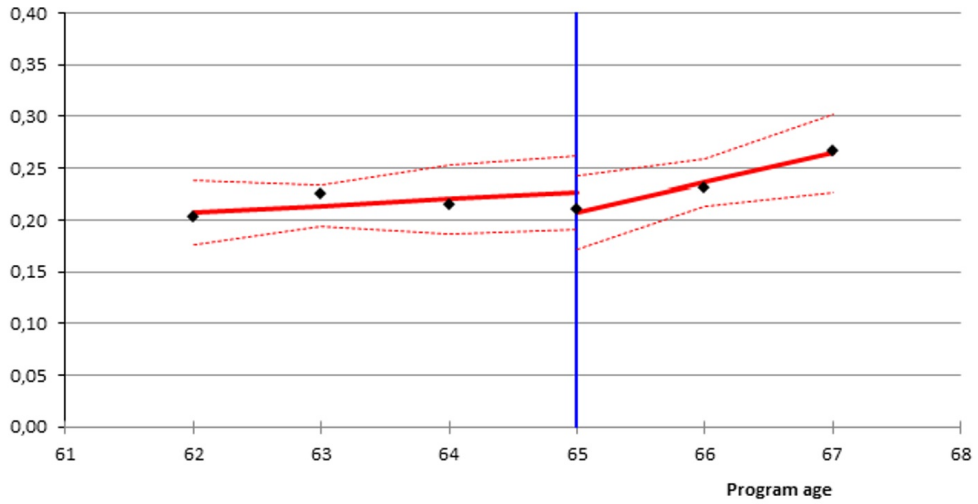
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the weighted average of individuals who visited a GP between Nov.-May, grouped in yearly bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.19: GP visited by adult children with parents around the age threshold



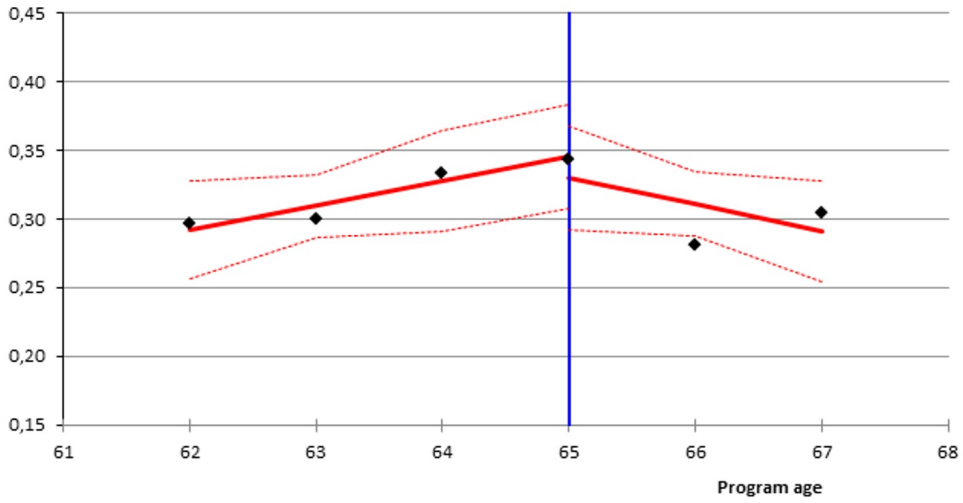
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average adult children who visited a GP between Nov.-May, grouped in yearly bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.20: Medical specialist visited by individuals around the 65-age threshold



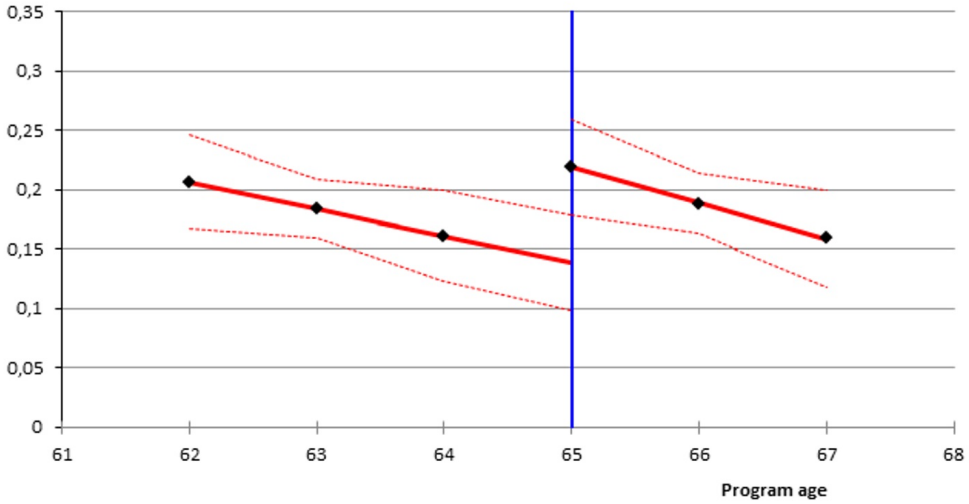
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the weighted average of individuals who visited a medical specialist between Nov.-May, grouped in yearly bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.21: Medical specialist visited by adult children with parents around the age threshold



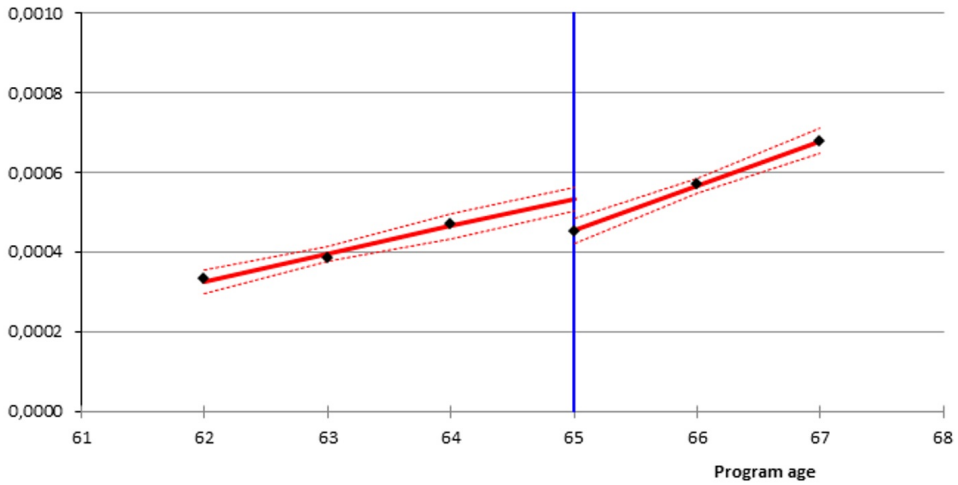
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average adult children who visited a medical specialist between Nov.-May, grouped in yearly bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.22: Sickness absence by adult children with parents around the age threshold



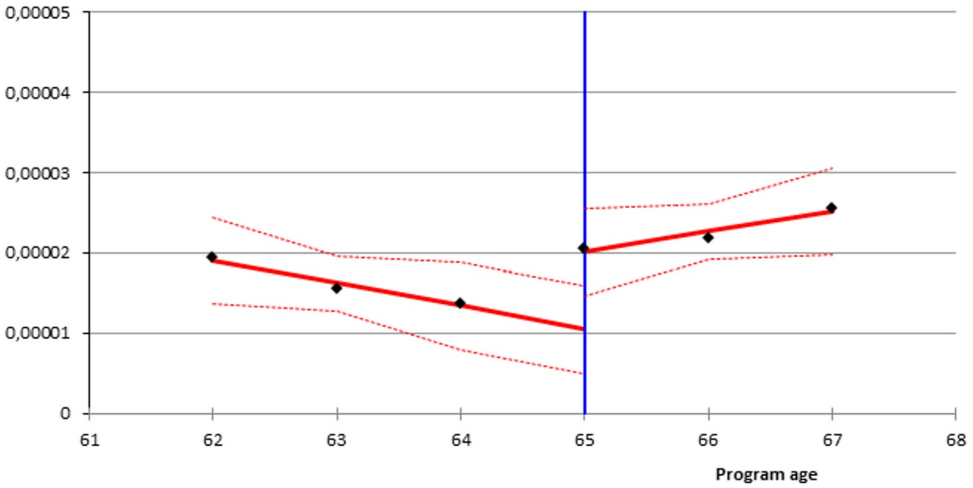
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the weighted average of sickness absence of adult children between Nov.-May, grouped in yearly bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.23: Mortality from influenza and pneumonia of individuals around the 65-age threshold



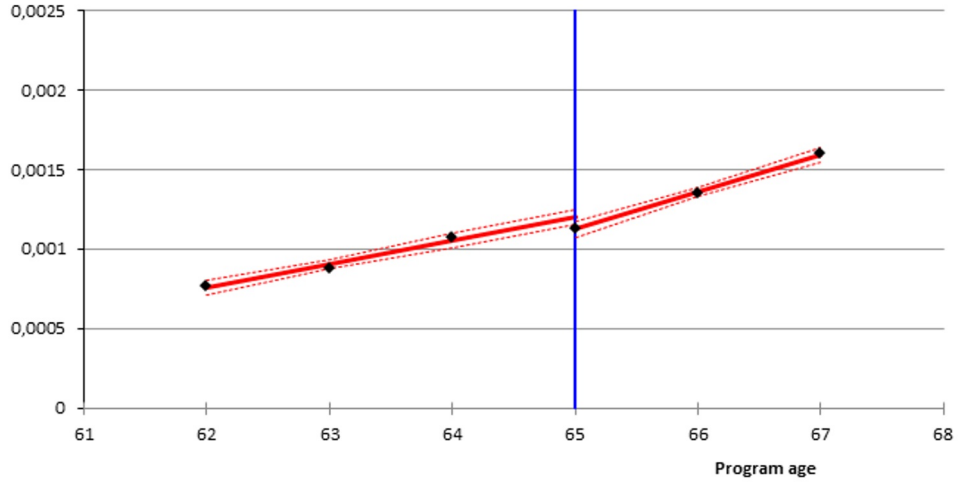
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the average mortality from influenza and pneumonia of individuals between Dec.-June, grouped in yearly bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.24: Mortality from influenza and pneumonia of adult children with parents around the age threshold



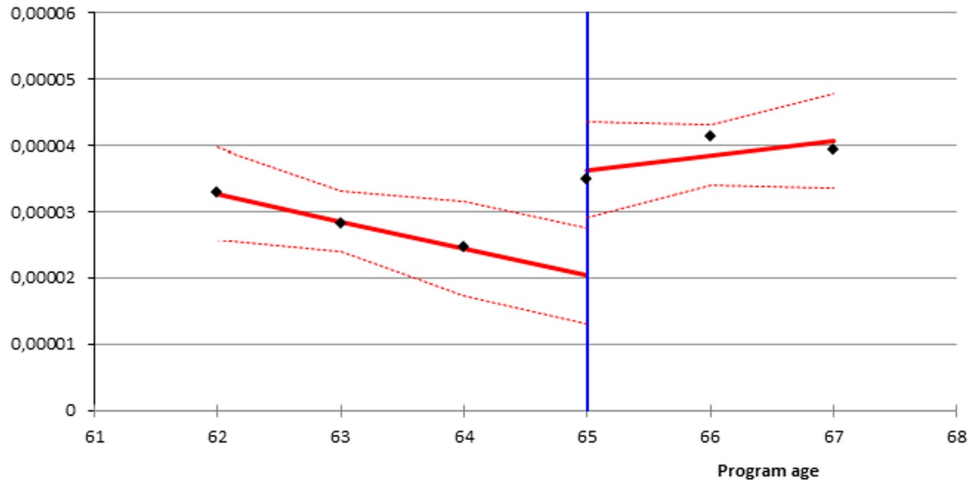
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the average mortality from influenza and pneumonia of adult children between Dec.-June, grouped in yearly bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.25: Mortality from respiratory diseases of individuals around the 65-age threshold



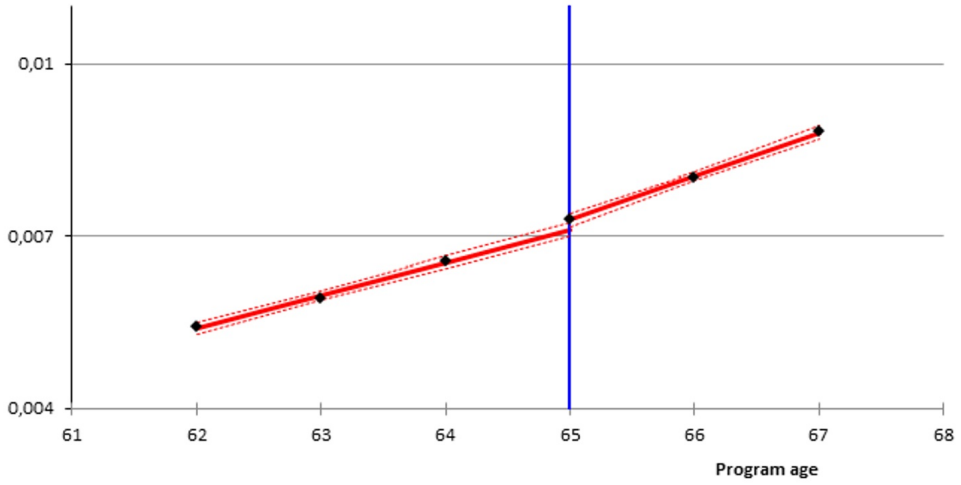
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the average mortality from respiratory diseases of individuals between Dec.-June, grouped in yearly bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.26: Mortality from respiratory diseases of adult children with parents around the age threshold



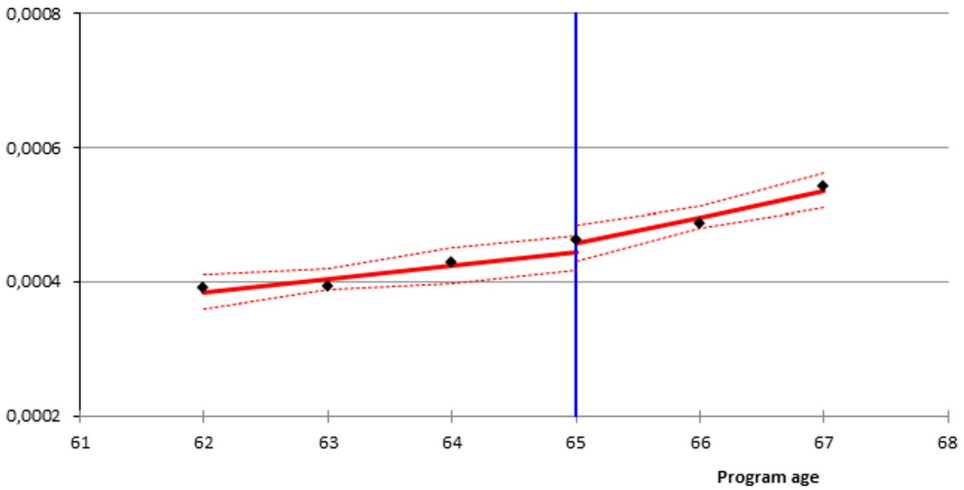
Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the average mortality from respiratory diseases of adult children between Dec.-June, grouped in yearly bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.27: All-cause mortality of individuals around the 65-age threshold



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the individual's program age. Each observation (represented by diamonds) is the average mortality of individuals between Dec.-June, grouped in yearly bins based on program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Figure 3.28: All-cause mortality of adult children with parents around the age threshold



Notes: Pooled data of the influenza seasons 1997-1998 to 2007-2008 are used. The running variable is the parent's program age. Each observation (represented by diamonds) is the average mortality of adult children between Dec.-June, grouped in yearly bins based on the oldest parent's program age. The solid line shows a linear trend based on the observations. The dotted lines represent the 95% confidence intervals. The vertical line represents the age threshold.

Table 3.8: RD estimates for other outcomes on parents and children during the influenza epidemic period (in a +/- 3 year window)

	Parents	**Children**
Influenza-like symptoms	0.047	0.077
	(0.051)	(0.050)
Non-prescribed medication	-0.044	-0.021
	(0.037)	(0.033)
Prescribed medication	-0.090	-0.028
	(0.031)***	(0.029)
GP visits	-0.090	-0.017
	(0.039)**	(0.030)
Visits to medical specialist	-0.024	0.003
	(0.035)	(0.033)
Sickness absence		0.076
		(0.035)**
Mortality: Pneumonia & influenza	-0.000063	0.000007
	(0.000028)**	(0.000005)
Mortality: respiratory diseases	-0.000050	0.000010
	(0.000043)	(0.000007)
Mortality: respiratory and circulatory diseases	-0.000013	0.000012
	(0.000074)	(0.000012)
Mortality: all-cause mortality	0.000139	-0.000002
	(0.000108)	(0.000024)

Notes: See text and Section 3.2 for details on the RD set-up. We use linear trends in program age that can differ at each side of the cutoff. Control variables include dummies for gender, member of risk group based on existing disorders, population density, chronic illness, education level, number of household members, family type and influenza season for outcomes 1 to 6. Control variables for mortality include gender and influenza season. In addition all regressions for children include dummies for children's program age per year and parent's gender. Standard errors between brackets. Regression estimates with HIS use sample weights and cluster standard errors at the wave-municipality level to mimic the sample design. Regressions on administrative data apply robust standard errors. The influenza epidemic period is defined as November-May: (a) influenza-like symptoms, sickness absence and GP/specialist visits refer to the last two months, so the survey months January to June are considered; (b) medicine use refers to the last month, so the survey months December to June are used; and (c) mortality refers to the actual months December-June. Significance level: * p<0.10, ** p<0.05, *** p<0.01.

Table 3.9: RD treatment estimates on covariates in equation (3.4)

	Sample with oldest parent's program age between 63 and 66	Sample with oldest parent's program age between 62 and 67
Male	-0.028 (0.040)	0.018 (0.029)
Program age	-0.039 (0.331)	0.040 (0.237)
Risk group	-0.026 (0.019)	-0.012 (0.013)
Chronic illness	0.013 (0.036)	-0.010 (0.025)
Education level 1 (primary school)	-0.003 (0.021)	-0.011 (0.016)
Education level 2	0.018 (0.031)	-0.006 (0.022)
Education level 3	-0.070 (0.040)	-0.032 (0.029)
Education level 4 (college education)	0.054 (0.037)	0.049 (0.026)
Number of household members	0.021 (0.052)	0.018 (0.039)
Family type: single person	-0.014 (0.030)	-0.030 (0.022)
Family type: couple	-0.049 (0.033)	-0.022 (0.025)
Family type: household with children	0.055 (0.073)	0.048 (0.027)
Family type: other	0.007 (0.006)	0.005 (0.004)
Oldest parent male	-0.039 (0.039)	-0.042 (0.028)
Population density: below 500 inhabitants/km^2	-0.031 (0.026)	-0.003 (0.019)
Population density: between 500 and 2500 inhabitants/km^2	0.007 (0.040)	-0.006 (0.030)
Population density: above 2500 inhabitants/km^2	0.024 (0.036)	0.009 (0.027)

Notes: See text and Section 3.2 for details on the RD set-up. We use linear trends in program age that can differ at each side of the cutoff. Sample restrictions and control variables are similar as those in Table 3.3 OLS regression estimates are reported that use sample weights and cluster standard errors at the wave-municipality level to mimic the sample design. Significance level: * p<0.10, ** p<0.05, *** p<0.01.

Table 3.10: RD treatment estimates on covariates in equation (3.2)

	Sample with program age between 63 and 66	Sample with program age between 62 and 67
Male	-0.034 (0.047)	-0.037 (0.046)
Risk group	0.020 (0.035)	0.016 (0.036)
Chronic illness	0.004 (0.042)	0.004 (0.042)
Education level 1 (primary school)	0.011 (0.035)	0.012 (0.035)
Education level 2	-0.024 (0.041)	-0.027 (0.041)
Education level 3	-0.036 (0.039)	-0.036 (0.039)
Education level 4 (college education)	0.049 (0.034)	0.050 (0.034)
Number of household members	0.017 (0.018)	0.017 (0.018)
Family type: single person	-0.065 (0.037)*	-0.067 (0.037)*
Family type: couple	0.099 (0.042)**	0.100 (0.042)**
Family type: household with children	-0.033 (0.026)	-0.033 (0.026)
Family type: other	-0.000 (0.009)	-0.000 (0.009)
Population density: below 500 inhabitants/kmš	-0.012 (0.030)	-0.013 (0.030)
Population density: between 500 and 2500 inhabitants/kmš	0.079 (0.042)*	0.075 (0.041)*
Population density: above 2500 inhabitants/kmš	-0.067 (0.035)*	-0.062 (0.033)*

Notes: See text and Section 3.2 for details on the RD set-up. We use linear trends in program age that can differ at each side of the cutoff. Sample restrictions and control variables are similar as those in Table 3.2 OLS regression estimates are reported that use sample weights and cluster standard errors at the wave-municipality level to mimic the sample design. Significance level: * p<0.10, ** p<0.05, *** p<0.01.

Table 3.11: Window robustness checks

	Parents	**Children**
+/- 1 year window	0.197***	-0.027**
	(0.022)	(0.013)
+/- 2 year window	0.168***	-0.040*
	(0.035)	(0.022)
+/- 3 year window	0.184***	-0.037**
	(0.026)	(0.016)
+/- 4 year window	0.216***	-0.026**
	(0.022)	(0.013)
+/- 5 year window	0.230***	-0.027**
	(0.020)	(0.011)
+/- 6 year window	0.242***	-0.027***
	(0.018)	(0.010)

Notes: See text and Section 3.2 for details on the RD set-up. We use linear trends in program age that can differ at each side of the cutoff. Sample restrictions and control variables are similar as those in Table 3.2 for parents and similar as those in Table 3.3 for adult children. OLS regression estimates are reported that use sample weights and cluster standard errors at the wave-municipality level to mimic the sample design. Standard errors between parentheses. Significance level: * p<0.10, ** p<0.05, *** p<0.01.

Bibliography

[1] Adato M. 2000. *The impact of PROGRESA on community social relationships.* Final Report PROGRESA, IFPRI: pp. 36.

[2] Adato M, de la Brière B, Mindek D, Quisumbing A 2000. *The impact of PROGRESA on women's status and intrahousehold relations.* Final Report PROGRESA, IFPRI: pp. 94.

[3] Agurto I, Bishop A, Sanchez G, Betancourt Z, Robles S. 2004. Perceived barriers and benefits to cervical cancer screening in Latin America. *Preventive Medicine*, 39, no. 1: pp. 91-98.

[4] Aizer A, Currie J. 2004. Networks or neighborhoods? Correlations in the use of publicly-funded maternity care in California. *Journal of Public Economics*, 88, no. 12: pp. 2573-2585.

[5] Akerlof, G. 1997. Social distance and Social Decisions. *Econometrica*, 65, no. 5: pp. 1005-1027.

[6] American Cancer Society. 2013. site: http://www.cancer.org/cancer/breastcancer/detailedguide/breast-cancer-survival-by-stage. Accessed on 02/12/2013.

[7] Angelucci M, De Giorgi G. 2009. Indirect Effects of an Aid Program: How Do Cash Transfers Affect Ineligibles' Consumption?. *American Economic Review*, 99, no. 1: pp. 486-508.

[8] Angelucci M, De Giorgi G., Rangel M A, Rasul I. 2010. Family networks and school enrolment: Evidence from a randomized social experiment. *Journal of Public Economics*, 94, no. 3: pp. 197-221.

[9] Angrist J D, Krueger A B. 1992. The Effect of Age at School Entry on Educational Attainment: An Application of Instrumental Variables with Moments from Two Samples. *Journal of the American Statistical Association*, 87, no. 418: pp. 328–36.

[10] Apouey B, Picone G. 2014. Social interactions and malaria preventive behaviors in sub-saharan Africa. *Health Economics*, online version

[11] Armendáriz de Aghion B, Morduch J. 2005. *The economics of microfinance.* The MIT Press, Cambridge.

[12] Auld M C. 2011. Effect of large-scale social interactions on body weight. *Journal of health economics*, 30, no. 2: pp. 303-316.

[13] Avitabile C. 2011. *Spillover effects in healthcare programs: Evidence on social norms and information sharing.* CSEF working paper no. 271: pp. 55.

[14] Bach P B, Mirkin J N, Oliver T K, Azzoli C G, Berry D A, Brawley O W, ... , Detterbeck F C. 2012. Benefits and harms of CT screening for lung cancer: a systematic review. *Jama*, 307, no. 22: pp. 2418-2429.

[15] Baird S, Bohren A, McIntosh C, Özler B. 2012. *Designing experiments to measure spillover and threshold effects*, Mimeo: pp. 61.

[16] Barham T. 2005. *The impact of the Mexican conditional cash transfer on immunization rates.* Mimeo, Department of Agriculture and Resource Economics, University of California at Berkeley.

[17] Barham T. 2011. A healthier start: the effect of conditional cash transfers on neonatal and infant mortality in rural Mexico. *Journal of Development Economics*, 94, no. 1: pp. 74-85.

[18] Barzallo D P. 2011. *Health conditions and social interactions.* IRENE working paper 11-03: pp. 33.

[19] Battistin E, Brugiavini A, Rettore E, Weber G. 2008. The Retirement Consumption Puzzle: Evidence from a Regression Discontinuity Approach. *The American Economic Review*, 99, no. 5: pp. 2209-2226.

[20] Behrman J R. 1996. The Impact of Health and Nutrition on Education. *World Rank Research Observer*, 11, no. 1: pp. 23-37.

[21] Behrman J R, Todd P. 1999. *Randomness in the Experimental Samples of PROGRESA.* Mimeo, IFPRI: pp. 36.

[22] Belkar R, Fiebig D, Haas M, Viney R. 2006. Why worry about awareness in choice problems? Econometric analysis of screening for cervical cancer. *Health Economics* 15: 33-47.

[23] Bish A, Yardley L, Nicoll A, Michie S. 2011. Factors associated with uptake of vaccination against pandemic influenza: A systematic review. *Vaccine*, 29: pp. 6472-6484.

[24] Blackwell D L, Lucas J W, Clarke T C. 2014. Summary health statistics for U.S. adults: National Health Interview Survey, 2012. National Center for Health Statistics. *Vital Health Statistics,* 10, no. 260.

[25] Blume L E, Brock W A, Durlauf S N, Ioannides Y M. 2010. Identification of Social Interactions (chapter 18, pp. 853-964). In: Benhabib J, Bisin A, and Jackson M. *Handbook of Social Economics*. North-Holland.

[26] Bobba M. and J. Gignoux (2012). *Policy-induced social interaction*s. Discussion paper: pp. 30.

[27] Bobonis G J. 2004. *Income transfers, marital dissolution, and intra-household resource allocation: Evidence from rural Mexico.* University of California at Berkeley working paper: pp. 52.

[28] Bobonis G J, Finan F. 2009. Neighborhood peer effects in secondary school enrolment decisions. *The Review of Economics and Statistics*, 91, no. 4: pp. 695-716.

[29] Börsch-Supan A, et al. 2005. *Health, ageing and retirement in Europe.* Mannheim: Research Institute for the Economics of Aging.

[30] Bouckaert N. 2014. *Neighborhood peer effects in the use of preventive health care.* CES-Discussion paper series, DPS14.03: pp. 47.

[31] Bruine de Bruin W, Fischbeck P S, Stiber N A, Fischhoff B. 2002. What Number is "Fifty-Fifty"?: Redistributing Excessive 50% Responses in Elicited Probabilities. *Risk Analysis*, 22, no.4: pp. 713-723.

[32] Buddelmeyer H, Skoufias E. 2004. *An Evaluation of the Performance of Regression Discontinuity Design on PROGRESA*. World Bank Policy Research Working Paper 3386.

[33] Bults M, Beaujean D J, Zwart O D, Kok G, Empelen P V, Steenbergen J V. 2010. Mexicaanse griep: risicoperceptie bij de bevolking, eigen maatregelen en vertrouwen in overheidsinformatie. *Ned Tijdschr Geneeskd*, 154, A1686.

[34] Byrne M, Thompson P. 2001. Screening and preventable illness. *Journal of Health Economics,* 20: pp. 1077-88.

[35] Card D, Dobkin C, Maestas N. 2008. The Impact of Nearly Universal Insurance Coverage on Health Care Utilization: Evidence from Medicare. *American Economic Review*, 98, no. 5: pp. 2242–58.

[36] Card D, Dobkin C, Maestas N. 2009. Does Medicare Save Lives? *Quarterly Journal of Economics*, 124, no. 2: pp. 597–636.

[37] Card D, Giuliano L. 2013. Peer effects and multiple equilibria in the risky behavior of friends. *Review of Economics and Statistics*, 95, no. 4: pp. 1130-1149.

[38] Carman K G., Kooreman P. 2010. Social interactions and obesity: An economist's perspective. (chapter 60, pp. 757-765). In Dube L., Bechara A., Dagher A., Drewnowski A., LeBel J., James P., Yada R.Y. (Eds.). *Obesity Prevention: The Role of Society and Brain on Individual Behavior*. Amsterdam: Elsevier.

[39] Carman K G, Kooreman P. 2011. *Flu shots, mammograms, and the perception of probabilities*. IZA: DP No. 5739.

[40] Carman K G, Mosca I. 2011. *Who Takes Advantage of Free Flu Shots? Examining the Effects Of An Expansion in Coverage*. Research Paper TILEC DP 2011-011.

[41] Carpenter C, Dobkin C. 2009. The Effect of Alcohol Consumption on Mortality: Regression Discontinuity Evidence from the Minimum Drinking Age.*American Economic Journal: Applied Economics*, 1, no. 1: pp. 164-182.

[42] Carrell S E, Hoekstra M, West J E. 2011. Is poor fitness contagious?: Evidence from randomly assigned friends. *Journal of Public Economics*, 95, no. 7: pp. 657-663.

[43] Case A, Fertig A, Paxson C. 2005. The Lasting Impact of Childhood Health and Circumstance. *Journal of Health Economics*, 24, no. 2: pp. 365-389.

[44] Case A, Lubotsky D, Paxson C. 2002. Economic Status and Health in Childhood: The Origins of the Gradient. *American Economic Review*, 92, no. 5: pp. 1308-1334.

[45] Case A, Paxson C. 2006. *Stature and Status: Height, Ability, and Labor Market Outcomes*. NBER working paper, no. w12466.

[46] CBS. 2003. *Vademecum gezondheidsstatistiek Nederland 2003.* Voorburg/Heerlen: pp. 260. http://www.cbs.nl/nr/rdonlyres/77848968-ee71-4c67-83a0-47dee6a316d4/0/ vademecum2003.pdf

[47] CDC. 2010.*Prevention and Control of Influenza with Vaccines. Recommendations of the Advisory Committee on Immunization Practices (ACIP).* Recommendations and Reports: 1-62. http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5908a1.htm [accessed 01/05/2014]

[48] CDC. 2014. *Fluview: a weekly influenza surveillance report prepared by the influenza division.* Centers for Disease Control and prevention. http://www.cdc.gov/flu/weekly/

[49] Chapman G B, Coups E J. 1999. Predictors of Influenza Vaccine Acceptance among Healthy Adults. *Preventive Medicine*, 29: pp. 249–262.

[50] Christakis N A, Fowler J H. 2007. The spread of obesity in a large social network over 32 years.*New England journal of medicine*, 357, no. 4: pp. 370-379.

[51] Christakis N A, Fowler J H. 2008. The collective dynamics of smoking in a large social network. *New England journal of medicine*, 358, no. 21: pp. 2249-2258.

[52] Cohen-Cole E, Fletcher J M. 2008a. Is obesity contagious? Social networks vs. environmental factors in the obesity epidemic. *Journal of Health Economics*, 27, no. 5: pp. 1382-1387.

[53] Cohen-Cole E, Fletcher J M. 2008b. Detecting implausible social network effects in acne, height, and headaches: longitudinal analysis.*British Medical Journal*, 337.

[54] Currie J. 2009. Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development. *Journal of Economic Literature*, 47, no. 1: pp. 87-122.

[55] Currie J, Madrian B C. 1999. Health, Health Insurance and the Labor Market. (pp. 3309-3416). In: Ashenfelter O, Card D. *Handbook of Labor Economics*, Volume 3C. Amsterdam; New York and Oxford: Elsevier Science, North-Holland.

[56] Cutler D, Deaton A, Lleras-Muney A. 2006. The Determinants of Mortality. *Journal of Economic Perspectives*, 20, no. 3: pp 97-120.

[57] Dahl G B, Løken K V, Mogstad M. 2014. Peer Effects in Program Participation. *American Economic Review*, 104, no. 7: pp. 2049-2074.

[58] de Bakker D, Jabaaij L, Abrahamse H, Hoogen van den H, Braspenning J, Althuis van T, Rutten, R. 2001. *Jaarrapport LINH 2000. Contactfrequenties, verrichtingen en monitoring griepvaccinatie en cervixscreening in het Landelijk Informatie Netwerk Huisartsenzorg (LINH).*Nivel.

[59] de la Mata D. 2011. *Estimating a dynamic discrete choice model of health prevention decisions: an application to flu vaccination.* Universidad Carlos III de Madrid: Mimeo.

[60] Deri C. 2005. Social networks and health service utilization. *Journal of Health Economics,* 24, no. 6: pp. 1076-1107.

[61] Dervaux B, Eeckhoudt L. 2004. Prévention en économie et en médecine. *Revue Economique,* 55, no. 5: pp. 849-56.

[62] Djulbegovic M, Beyth R J, Neuberger M M, Stoffs T L, Vieweg J, Djulbegovic B, Dahm P. 2010. Screening for prostate cancer: systematic review and meta-analysis of randomised controlled trials. *BMJ: British Medical Journal*, 341.

[63] Donker G. 2010. *Continue Morbiditeits Registratie Peilstations Nederland 2009.* Nivel.

[64] Duncan G J, Boisjoly J, Kremer M, Levy D M, Eccles J. 2005. Peer effects in drug use and sex among college students.*Journal of abnormal child psychology,* 33, no. 3: pp. 375-385.

[65] Etner J, Jeleva M. 2013. Risk perception, prevention and diagnostic tests. *Health Economics, 22*: pp. 144-156.

[66] European Commission (2014). *State of play on implementation of the Council Recommendation of 22 December 2009 on seasonal influenza vaccination (2009/1019/EU).* Brussels, http://ec.europa.eu/health/vaccination/docs/seasonflu_staffwd2014_en.pdf

[67] Fang H, Wang Y. 2010. *Estimating dynamic discrete choice models with hyperbolic discounting, with an application to mammography decisions.* NBER: Working Paper no. 16438.

[68] Fedson D S, Hirota Y, Shin H K, Cambillard P E, Kiely J, Ambrosch F, ... , Strikas R A. 1997. Influenza vaccination in 22 developed countries: an update to 1995. *Vaccine*, 15, no. 14: pp. 1506-1511.

[69] Fedson D S, Hannoun C, Leese J, Sprenger M J, Hampson A W, Bro-Jørgensen K, ... , Donatelli I. 1995. Influenza vaccination in 18 developed countries, 1980–1992. *Vaccine*, 13, no. 7: pp. 623-627.

[70] Fels M. 2011. *The value of medical diagnosis: why people reject medical information.* University of Bonn: Mimeo.

[71] Finkelstein A, Luttmer E F P, Notowidigdo M J. 2009. Approaches to Estimating the Health State Dependence of the Utility Function. *American Economic Review,* 99: pp. 116-121.

[72] Fletcher J M. 2010. Social interactions and smoking: Evidence using multiple student cohorts, instrumental variables, and school fixed effects. *Health Economics*, 19, no. 4: pp. 466-484.

[73] Fletcher J M. 2012. Peer influences on adolescent alcohol consumption: evidence using an instrumental variables/fixed effect approach.*Journal of Population Economics*, 25, no. 4: pp. 1265-1286.

[74] Forsyth K D, Wirsing von Konig C H, Tan T, Caro J, Plotkin S. 2007. Prevention of pertussis: recommendations derived from the second Global Pertussis Initiative roundtable meeting. *Vaccine*, 25, no. 14: pp. 2634-2642.

[75] Fowler J H, Christakis N A. 2008. Estimating peer effects on health in social networks: A response to Cohen-Cole and Fletcher; Trogdon, Nonnemaker, Pais.*Journal of Health Economics*, 27, no. 5: pp. 1400-1405.

[76] Galvani A P, Reluga T C, Chapman G B. 2007. Long-standing influenza vaccination policy is in accord with individual self-interest but not with the utilitarian optimum. *Proceedings of the National Academy of Sciences,*104, no. 13: pp. 5692-5697.

[77] Gertler P. 2000. *The impact of PROGRESA on health.* Final Report PROGRESA, IFPRI: pp. 31.

[78] Gertler P. 2004. Do Conditional Cash Transfers Improve Child Health? Evidence from PROGRESA's Control Randomized Experiment. *American Economic Review*, 94, no. 2: pp. 336-341.

[79] Godlonton S, Thornton R. 2012. Peer effects in learning HIV results. *Journal of development economics*, 97, no. 1: pp. 118-129.

[80] Gray N. 2013.*Social Interactions and Breast Cancer Prevention among Women over Age 50 in the US.* Mimeo

[81] Grohskopf L, Shay D, Shimabukuro T, Sokolow L, Keitel W, Bresee J, Cox N. 2013. *Prevention and Control of Seasonal Influenza with Vaccines: Recommendations of the Advisory Committee on Immunization Practices — United States, 2013–2014.* Recommendations and Reports September 20, 2013 / 62(RR07): pp. 43.

[82] Hadler J L, Konty K, McVeigh K H, Fine A, Eisenhower D, Kerker B, Thorpe L. 2010. Case fatality rates based on population estimates of influenza-like illness due to novel H1N1 influenza: New-York City. *PloS One,* 5, no. 7.

[83] Hak E, Hermens R, Hoes ., Verheij ., Kuyvenhoven ., van Essen G. 2000. Effectiveness of a co-ordinated nation-wide programme to improve influenza immunisation rates in The Netherlands.*Scand J Prim Health Care,* 18: pp. 237-241.

[84] Halliday T J, Kwak S. 2009. Weight gain in adolescents and their peers. *Economics & Human Biology,*7, no. 2, pp. 181-190.

[85] Halloran M E, Longini Jr. I M. 2006. Community Studies for Vaccinating Schoolchildren Against Influenza. *Science*, 311: pp. 615-616.

[86] Hoddinott J, Skoufias E. 2004. The Impact of PROGRESA on Food Consumption. *Economic development and cultural change*, 53, no. 1: pp. 37-61.

[87] Howard D. 2005. Life expectancy and the value of early detection. *Journal of Health Economics,* 24: pp. 891-906.

[88] Imbens G W, Kalyanaraman K. 2012. Optimal Bandwidth Choice for the Regression Discontinuity Estimator. *Review of Economic Studies*, 79, no. 3: pp. 933-959.

[89] INSP. 2005. *General Rural Methodology Note.* Instituto Nacional de Salud Pública (INSP), Sedesol.

[90] Jansen B, Tacken M, Mulder J, Korevaar J, Schlief A, Tiersma W, Braspenning J. 2013. *LINH Monitoring Vaccinatiegraad Nationaal Programma Griepppreventie 2012.* LINH.

[91] Jordan R, Connock M, Albon E, Fry-Smith A, Olowokure B, Hawker J, Burls A. 2006. Universal vaccination of children against influenza: are there indirect benefits to the community?: a systematic review of the evidence. *Vaccine* 24, no. 8: pp. 1047-1062.

[92] Juckett D A, Rosenberg B. 1993. Comparison of the Gompertz and Weibull functions as descriptors for human mortality distributions and their intersections. *Mechanisms of ageing and development*, 69, no. 1: pp. 1-31.

[93] Jusot F, Or Z, Sirven N. 2012. Variations in preventive care utilisation in Europe. *European Journal of Ageing,* 9: pp. 15-25.

[94] Kremer M, Levy D. 2008. Peer effects and alcohol use among college students. *Journal of Economic Perspectives*, 22, no. 3: pp. 189-206.

[95] Kremer M, Miguel E. 2007. The illusion of sustainability. *The Quarterly Journal of Economics*, 122, no. 3: pp. 1007-1065.

[96] Kroneman M W, Verheij R. 2003. *De griepprik in Nederland: motivatie voor deelname en distributiekanalen.* NIVEL rapport.

[97] Lagarde M, Haines A, Palmer N. 2007. Conditional cash transfers for improving uptake of health interventions in low-and middle-income countries. *JAMA: the journal of the American Medical Association*, 298, no. 16: pp. 1900-1910.

[98] Lalive R, Cattaneo A. 2009. Social interactions and schooling decisions. *The Review of Economics and Statistics*, 91, no. 3: pp. 457-477.

[99] Lee D, Lemieux T. 2010. Regression Discontinuity Designs in Economics. *Journal of Economic Literature*, 48: pp. 281–355.

[100] Lemieux T, Milligan K. 2008. Incentive effects of social assistance: A regression discontinuity approach. *Journal of Econometrics*, 142, no. 2: pp. 807-828.

[101] Lewis M J. 2004. *A Situational Analysis of Cervical Cancer in Latin America and the Caribbean.* PAHO, Washington, DC : pp. 29.

[102] Listl S. 2011. Income-related inequalities in dental service utilization by Europeans aged 50+. *Journal of Dental Research,* 90, no. 6: pp. 717-723.

[103] Listl S, Moran V, Maurer J, Faggion Jr C M. 2012. Dental service utilization by Europeans aged 50 plus. *Community Dentistry and Oral Epidemiology,* 40: 164-174.

[104] Manski C F. 1993. Identification of endogenous social effects: The reflection problem. *The Review of Economic Studies*, 60, no. 3: pp. 531-542.

[105] Marmot M G, Altman D G, Cameron D A, Dewar J A, Thompson S G, Wilcox M. 2013. The benefits and harms of breast cancer screening: an independent review. *British journal of cancer*, 108, no. 11: pp. 2205-2240.

[106] Maurer J. 2009. Who has a clue to preventing the flu? Unravelling supply and demand effects on the take-up of influenza vaccinations. *Journal of Health Economics,* 28: pp. 704-17.

[107] Meerhoff T, Paget W, Aguilera J, van der Velden J. 2004. Harmonising the virological surveillance of influenza in Europe: results of an 18-country survey. *Virus research*, 103: pp. 31-33.

[108] Mereckiene J, Cotter S, Nicoll A, Lopalco P, Noori T, Weber J T., ... , O'Flanagan D. 2014. Seasonal influenza immunisation in Europe. Overview of recommendations and vaccination coverage for three seasons: pre-pandemic (2008/09), pandemic (2009/10) and post-pandemic (2010/11). *Eurosurveillance*19, no. 16.

[109] Mereckiene J, Cotter S, Weber J T, Nicoll A, D'Ancona F, Lopalco P L, Johansen K, Wasley A M, Jorgensen P, Lévy-Bruh D, Giambi C, Stefanoff P, Dematte L, O'Flanagan D. 2012. The VENICE project gatekeepers group. *Eurosurveillance,* 17, no. 4.

[110] Meredith J M, Robinson J, Walker S, Wydick B. 2013. *Keeping the doctor away: experimental evidence on investment in preventative health products.* NBER working paper, no. w19312.

[111] Moffitt R A. 1983. An economic model of welfare stigma, *American Economic Review*, 73, no. 5: pp. 1023-1035.

[112] Moffitt R A. 2001. Policy Interventions, Low Level Equilibria, and Social Interactions (chapter 3, pp. 45–82). In Durlauf, S.N. and H. P. Young. *Social Dynamic*s. The MIT Press.

[113] Mossialos E, Thomson S. 2002. Voluntary health insurance in the European Union (chapter 6, pp. 128-160). In Mossialos E, Dixon A, Figueras J, Kutzin J. *Funding health care in: options for Europe.* Open University Press.

[114] Mullahy J. 1999. It'll only hurt a second? Microeconomic determinants of who gets flu shots. *Health Economics,* 8: pp. 9-24.

[115] Munshi K, Myaux J. 2006. Social norms and the fertility transition. *Journal of Development Economics*, 80, no. 1: pp. 1-38.

[116] Nagata J M, Hernandez-Ramos I, Kurup A S, Albrecht D, Vivas-Torrealbas C, Franco-Paredes C. 2011. Social determinants of health and seasonal influenza vaccination in

adults≥65 years: a systematic review of qualitative and quantitative data. *BMC Public Health*, 13.

[117] National Center for Health Statistics. 2012. *Healthy People 2010 Final Review.* Hyattsville, MD.

[118] National Center for Health Statistics. 2014. *Health, United States, 2013: With Special Feature on Prescription Drugs.* DHHS Publication no. 2014-1232.

[119] Nichol K L. 2003. The efficacy, effectiveness and cost-effectiveness of inactivated influenza virus vaccines. *Vaccine*, 21, no. 16: pp. 1769-1775.

[120] Nichol K. 2008. Efficacy and effectiveness of influenza vaccination. *Vaccine*, 26, supplement 4: pp. D17-D22.

[121] Nicoll A, Ciancio B C, Lopez Chavarrias V, Mølbak K, Pebody R, Pedzinski B, Penttinen P, van der Sande M, Snacken R, Van Kerkhove M. D. 2012. Influenza-related deaths–available methods for estimating numbers and detecting patterns for seasonal and pandemic influenza in Europe. *Eurosurveillance*, 17, no. 18: pp. 1-13.

[122] Noguera J A, Miguel F J, Tapia E, Llàcer T. 2013. *Tax compliance, rational choice and social influence: An agent based model.* Mimeo: pp. 33.

[123] Oakley Jr. G P, Johnston Jr. R B. 2004. Balancing benefits and harms in public health prevention programmes mandated by governments. *BMJ: British Medical Journal,* 329, no. 7456: pp. 41-43.

[124] OECD Health Data. http://stats.oecd.org/index.aspx?DataSetCode=HEALTH_STAT#

[125] O'Flanagan D, Cotter S, Mereckiene J. 2012. *Seasonal influenza vaccination in EU/EEA, influenza season 2010-2011.* Venice II consortium. http://venice.cineca.org/Final_Seasonal_Influenza_2010-11.pdf

[126] Oster E, Shoulson I, Dorsey R. 2011. *Optimal expectations and limited medical testing: evidence from Huntington disease.* NBER: Working Paper no. 17629.

[127] Oster E, Thornton R. 2012. Determinants of technology adoption: Peer effects in menstrual cup take-up. *Journal of the European Economic Association*, 10, no. 6: pp. 1263-1293.

[128] Peracchi F, Perotti V. 2011. *Subjective survival probabilities and life tables: evidence from Europe.* Mimeo.

[129] Picone G, Sloan F, Taylor D. 2004. Effects of risk and time preference and expected longevity on demand for medical tests. *Journal of Risk and Uncertainty,* 28, no. 1: pp. 39-53.

[130] PROGRESA. http://www.oportunidades.gob.mx:8010/index1.php

[131] Ranganathan M, Lagarde M. 2012. Promoting healthy behaviors and improving health outcomes in low and middle income countries: A review of the impact of conditional cash transfer programmes. *Preventive Medicine*, 55: pp. S95-S105.

[132] Rao N, Kremer M, Mobius M, Rosenblat T. 2012. *Social Networks and Vaccination Decisions*. Mimeo

[133] Reade C J, Riva J J, Busse J W, Goldsmith C H, Elit L. 2013. Risks and benefits of screening asymptomatic women for ovarian cancer: a systematic review and meta-analysis. *Gynecol Oncol*, 130, no. 3: pp. 674-681.

[134] Reichert T A, Sugaya N, Fedson D S, Glezen W P, Simonsen L, Tashiro M. 2001. The Japanese experience with vaccinating schoolchildren against influenza. *New England Journal of Medicine*, 344, no. 12: pp. 889-896.

[135] RIVM. 2014. [Accessed on 21/04/2014] . http://www.rivm.nl/Onderwerpen/G/Griepprik/Veelgestelde_vragen/veelgestelde_vragen/Hoe_kan_ik_een_griepprik_krijgen

[136] Roos A, Schut E. 2008. *Evaluatie aanvullende en collectieve verzekeringen 2008. Onderzoek in opdracht van de Nederlandse Patiënten Consumenten Federatie.*Erasmus University Rotterdam.

[137] Rosenquist J N, Murabito J, Fowler J H, Christakis N A. 2010. The spread of alcohol consumption behavior in a large social network. *Annals of Internal Medicine*, 152, no. 7: pp. 426-433.

[138] Sánchez-Castillo C P, Velásquez-Monroy O, Lara-Esqueda A, Berber A, Sepúlveda J, Tapia-Conyer R, James W P T. 2005. Diabetes and hypertension increases in a society with abdominal obesity: results of the Mexican National Health Survey 2000. *Public health nutrition*, 8, no. 1: pp. 53-60.

[139] Schmitz, H, Wübker A. 2011. What determines influenza vaccination take-up of elderly Europeans? *Health Economics,* 20: pp. 1281-1297.

[140] Sepúlveda J, Bustreo F, Tapia R, Rivera J, Lozano R, Oláiz G, Partida V, Garciá-Garciá L, Valdespino J L. 2006. Improvement of child survival in Mexico: the diagonal approach. *The Lancet*, 368: pp. 2017-2027.

[141] SHARE. http://www.share-project.org/

[142] Skoufias E. 2005. *PROGRESA and its impacts on the welfare of rural households in Mexico.* Research Report PROGRESA, no. 139, IFPRI: pp. 84.

[143] Smith R A, Cokkinides V, Brooks D, Saslow D, Brawley O W. 2010. Cancer Screening in the United States, 2010: A Review of Current American Cancer Society Guidelines and Issues in Cancer Screening. *CA: A Cancer Journal for Clinicians*, 60: pp. 99–119.

[144] Smith V, Taylor D, Sloan F. 2001. Longevity expectations and death: can people predict their own demise? *American Economic Review,* 91, no. 4: pp. 1126-34.

[145] Stancanelli E, van Soest A. 2012. Retirement and Home Production: A Regression Discontinuity Approach. *The American Economic Review*, 102, no. 3: pp. 600-605.

[146] Stock J H, Yogo M. 2005. Testing for Weak Instruments in Linear IV Regression. (pp. 109–120). In: Andrews D, Stock J H. *Identification and Inference for Econometric Models: Essays in Honor of Thomas Rothenberg.* Cambridge University Press.

[147] Tacken M, de Bakker D, Verheij R, Mulder J, Hoogen van den H, Braspenning J. 2002.*LINH Evaluatie Griepvaccinatiecampagne 2001.* Nivel.

[148] Tacken M, Berende A, Verheij R, Mulder J, Hoogen van den H, Braspenning J. 2003. *LINH Evaluatie Griepvaccinatiecampagne 2002.* Nivel.

[149] Tacken M, Verheij R, Mulder J, Hoogen van den H, Braspenning J. 2004. *LINH Monitoring Griepvaccinatiecampagne 2003.* Nivel.

[150] Tacken M, Verheij R, Mulder J, Hoogen van den H, Braspenning J. 2005. *LINH Monitoring Griepvaccinatiecampagne 2004.* Nivel.

[151] Tacken M, Verheij R, Mulder J, Hoogen van den H, Braspenning J. 2006. *LINH Monitoring Griepvaccinatiecampagne 2005.* Nivel.

[152] Tacken M, Mulder J, Hoogen van den H, Donkers J, Tiersma W, Verheij R, Braspenning J. 2007. *LINH Monitoring Griepvaccinatiecampagne 2006.* Nivel.

[153] Tacken M, Mulder J, Hoogen van den H, Tiersma W, Verheij R, Braspenning J. 2008. *LINH Monitoring Nationaal Programma Griepppreventie 2007.* LINH.

[154] Tacken M, Mulder J, Hoogen van den H, Tiersma W, Donkers J, Verheij R, Braspenning J. 2009. *LINH Monitoring Nationaal Programma Griepppreventie 2008.* LINH.

[155] Tacken M, Mulder J, Hoogen van den H, Tiersma W, Donkers J, Verheij R, Braspenning J. 2010. *LINH Monitoring Vaccinatiegraad Nationaal Programma Griepppreventie 2009.* LINH.

[156] Thomas R E, Russell M L, Lorenzetti D L. 2010. Systematic review of interventions to increase influenza vaccination rates of those 60 years and older. *Vaccine*, 28, no. 7: pp. 1684-1701.

[157] Thomas R, Jefferson T, Demicheli V, Rivetti D. 2006. Influenza vaccination for health-care workers who work with elderly people in institutions: a systematic review. *Lancet Infectious Diseases*, 6, no. 5: pp. 273-279.

[158] Thompson M G, Shay D K, Zhou H, Bridges C B, Cheng P Y, Burns E, Bresee J S, Cox N J. 2010. Updated estimates of mortality associated with seasonal influenza – United States, 1976 – 2007.*Morbidity and Mortality Weekly Report* 59, no. 33: pp. 1057-1062.

[159] Trogdon J G, Nonnemaker J, Pais J. 2008. Peer effects in adolescent overweight. *Journal of Health Economics*, 27, nor. 5: pp. 1388-1399.

[160] Van den Berg G J, Lindeboom M, Portrait F. 2006. Economic Conditions Early in Life and Individual Mortality. *American Economic Review*, 96, no. 1: pp. 290-302.

[161] van der Weerd W, Timmermans D R, Beaujean D J, Oudhoff J, van Steenbergen J E. 2011. Monitoring the level of government trust, risk perception and intention of the general public to adopt protective measures during the influenza A (H1N1) pandemic in the Netherlands. *BMC public health*, 11, no. 1: pp. 575-587.

[162] van Doorslaer E, Masseria C, Koolman X. 2006. Inequalities in access to medical care by income in developed countries. *Canadian Medical Association Journal*, 174, no. 2: pp. 177-183.

[163] van Essen G A, Kuyvenhoven M M, de Melker R A. 1997. Compliance with influenza vaccination. Its relation with epidemiologic and sociopsychological factors. *Arch. Fam Med,*6: pp. 157-162.

[164] van Essen G A, Kuyvenhoven M M, Masurel N, Diepersloot R J A, van der Graaf M, Verheij T J. 2001. Influenza vaccination in the Netherlands: a successful system approach. *International Congress Series,* 1219: pp. 689-695.

[165] VENICE. http://venice.cineca.org/

[166] Viboud C, Bjørnstad O N, Smith D L, Simonsen L, Miller M A, Grenfell B T. 2006. Synchrony, Waves, and Spatial Hierarchies in the Spread of Influenza. *Science*, 312: pp. 447-451.

[167] Viscusi W K, Hakes J. 2003. Risk ratings that do not measure probabilities. *Journal of Risk Research*, 6, no. 1: pp. 23-43.

[168] von Karsa L, Anttila A, Ronco G, Ponti A, Malila N, Arbyn M, Segnan N, Castillo-Beltran M, Boniol M, Ferlay J, Hery C, Sauvaget C, Voti L, Autier P. 2008. *Cancer Screening in the European Union. Report on the implementation of the Council Recommendation on cancer screening.* European Communities.

[169] Ward C J. 2014. Influenza Vaccination Campaigns: Is an Ounce of Prevention Worth a Pound of Cure?. *American Economic Journal: Applied Economics*, 6, nor. 1: pp. 38-72.

[170] Watkins M M, Gabali C, Winkleby M, Gaona E, Lebaron S. 2002. Barriers to cervical cancer screening in rural Mexico. *International journal of Gynecological cancer*, 12, no. 5: pp. 475-479.

[171] Weon B M. 2004. *Analysis of trends in human longevity by new model.* Mimeo.

[172] WHO. 2005. Influenza vaccines. WHO position paper. *The Weekly Epidemiological Record*, 33: pp. 279-287.

[173] WHO. 2014. *Flunet.* http://www.who.int/influenza/gisrs_laboratory/flunet/charts/en/

[174] WHO GLOBOCAN. http://globocan.iarc.fr/Pages/DataSource_and_methods.aspx

[175] Whynes D, Philips Z, Avis M. 2007. Why do women participate in the English cervical cancer screening programme? *Journal of Health Economics,* 26: pp. 306-25.

[176] Widström E, Eaton K A. 2004. Oral healthcare systems in the extended European union. *Oral Health & Preventive Dentistry*, 2, no. 3: pp. 155-94.

[177] Wilson D L. 1994. The analysis of survival (mortality) data: fitting Gompertz, Weibull, and logistic functions. *Mechanisms of ageing and development.* 74, no.1: pp. 15-33.

[178] Witt J. 2008. The effect of information in the utilization of preventive health-care strategies: an application to breast cancer. *Health Economics,* 17: pp. 721-31.

[179] World Bank (2004). *Poverty in Mexico: An Assessment of Conditions, Trends and Government Strategy*. Report no. 28612-ME.

[180] World Bank (2005). *Mexico - Income Generation and Social Protection for the Poor: Volume 4. A Study of Rural Poverty in Mexico*. Report no. 32867-MX.

[181] Wu S. 2003. Sickness and preventive medical behavior. *Journal of Health Economics* 22: pp. 675-689.

[182] Wübker A. 2012a. *Explaining variations in breast cancer screening across European countries*. Ruhr Economic Papers, no. 370.

[183] Wübker A. 2012b. Who gets a mammogram amongst European women aged 50-69 years? *Health Economics Review* 2, no. 6: pp. 1-13.

[184] Yakusheva O, Kapinos K, Weiss M. 2011. Peer effects and the freshman 15: evidence from a natural experiment. *Economics & Human Biology,* 9, no. 2: pp. 119-132.

[185] Young H P. 2009. Innovation diffusion in heterogeneous populations: Contagion, social influence, and social learning. *American Economic Review*, 99, no. 5: pp. 1899-1924.

[186] Zijtregtop E A M, Wilschut J, Koelma N, Van Delden J J M, Stolk R P, Van Steenbergen J, Broer J, Wolters B, Postma M J, Hak E. 2010. Which factors are important in adults' uptake of a (pre)pandemic influenza vaccine? *Vaccine*, 28: pp. 207-227.

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